



11 E.



## LIBRARY

**Author:** HUMPHRY (G M )

**Title:** The collected investigation record.

**Acc No.**  
60334

**Classmark**  
EP

**Year**  
1883

**Volume**



Digitized by the Internet Archive  
in 2015

<https://archive.org/details/b24997730>







THE  
COLLECTIVE INVESTIGATION  
RECORD.

EDITED

*FOR THE COLLECTIVE INVESTIGATION COMMITTEE  
OF THE BRITISH MEDICAL ASSOCIATION*

BY

PROFESSOR HUMPHRY, M.D., F.R.S.,  
CHAIRMAN OF THE COMMITTEE,

AND

F. A. MAHOMED, M.B., F.R.C.P.,  
SECRETARY TO THE COMMITTEE.

---

JULY, 1883.

---

PRINTED AND PUBLISHED BY THE  
BRITISH MEDICAL ASSOCIATION, 161A, STRAND.

**DR. HENEAGE GIBBES'**  
**NEW DOUBLE STAIN FOR TUBERCLE BACILLUS,**  
 AND  
**OTHER STAINING FLUIDS FOR ALL BACTERIA,**  
**HISTOLOGICAL & PATHOLOGICAL WORK.**

*The most recent Formulæ. Directions for Use with each Bottle.*

**MOUNTED SPECIMENS OF BACTERIA.**

Microscope specially adapted for the investigation of above, £8 12s. 6d.

DETAILED LISTS ON APPLICATION TO

**R. & J. BECK, 68, CORNHILL, LONDON, E.C.**

*IN THE PRESS.*

**THE PHYSIOLOGICAL FACTOR IN DIAGNOSIS:**

A WORK FOR MEDICAL MEN COMMENCING PRACTICE.

By J. MILNER FOTHERGILL, M.D.

**WAUGH'S**

The attention of the Medical Profession is invited to this preparation, which represents the *Confectio Sennæ B.P.*, in a fluid state.

In Bottles of  
 4, 8, 12, and  
 24 Fluid ozs.,  
 or in Bulk at  
 3/6 per lb.

**FLUID CONFECTION**

See opinions of  
 the Medical  
 Press, and tes-  
 timony as to  
 its superiority  
 over the ordinary Pre-  
 parations of Senna.

The advantages clearly for this over the ordinary  
 fection are non-liability to fermentation even at a high  
 temperature, accuracy of measurement, and the removal  
 of the difficulty which many patients experience in  
 taking the old confection.

**OF SENNA.**

Extract from "Lancet," March 17th, 1883.

"In this preparation a palatable taste of Senna is remarkably well  
 masked. We think it a great improvement on the old Confection."

Extract from "British Medical Journal,"  
 June 30th, 1883.

"The Fluid Confection of Senna now made by Messrs. Waugh & Co., of  
 Regent Street, is a palatable preparation which will be found useful in the treat-  
 ment of habitual constipation. We have given it a fair trial, and find that it  
 is popular with patients."

**GEO. WAUGH & CO., 177, REGENT ST., W.**

# CONTENTS.

---

	PAGE
A SHORT HISTORY OF THE MOVEMENT. By Prof. HUMPHRY, M.D., F.R.S. . . . .	1
AN ADDRESS ON THE COLLECTIVE INVESTIGATION OF DIS- EASE. By Sir W. GULL, BART., PHYSICIAN EXTRAORDINARY TO H.M. THE QUEEN. . . . .	7
AN ADDRESS ON THE COLLECTIVE INVESTIGATION OF DIS- EASE. By Sir JAMES PAGET, BART., SERJEANT-SURGEON TO H.M. THE QUEEN . . . . .	21
A REPORT ON THE COMMUNICABILITY OF PHTHISIS . . . . .	26
APPENDIX OF RETURNS ON THE COMMUNICABILITY OF PHTHISIS . . . . .	43
A PRELIMINARY REPORT ON ACUTE PNEUMONIA . . . . .	93
A PRELIMINARY REPORT ON CHOREA . . . . .	111
A PRELIMINARY REPORT ON ACUTE RHEUMATISM . . . . .	119
A PRELIMINARY REPORT ON DIPHTHERIA . . . . .	128
THE SUBJECT FOR THE NEXT INQUIRY:—	
THE TREATMENT OF ACUTE GOUT . . . . .	134
MEMORANDUM AND CARD . . . . .	138
ORIGINAL COMMUNICATIONS:—	
A CALCULATION OF THE PROBABILITY OF THE ACCIDENTAL AND FATAL INCIDENCE OF PHTHISIS UPON BOTH HUSBAND AND WIFE. By G. B. LONGSTAFF, M.A., M.B. OXON., M.R.C.P. . . . .	139
OBSERVATIONS ON THE COLLECTIVE INVESTIGATION OF DISEASE, ESPECIALLY WITH REGARD TO ACUTE PNEUMONIA. By R. L. BOWLES, M.D., F.R.C.P. . . . .	145



# CONTENTS.

	PAGE
SUGGESTIONS FOR FUTURE INVESTIGATION :—	
INQUIRY INTO THE ASSOCIATION OF WHOOPING COUGH WITH MEASLES. BY W. B. CHEADLE, M.A., M.D. CANTAB., F.R.C.P., PHYSI- CIAN TO ST. MARY'S HOSPITAL, AND TO THE HOSPITAL FOR SICK CHILDREN, GREAT ORMOND STREET . . . . .	151
AN INQUIRY CARD ON WHOOPING COUGH. BY JUDSON S. BURY, M.D., B.S. LOND. . . . .	153
THERAPEUTICS. BY W. E. BUCK, M.A., M.D. CANTAB., PHYSICIAN TO THE LEICESTER INFIRMARY AND FEVER HOUSE . . . . .	154
AN INQUIRY ON INFECTIOUS SORE THROAT, WITHOUT RASH. BY E. T. WILSON, M.B. OXON., F.R.C.P., PHYSICIAN TO THE CHELTENHAM GENERAL HOSPITAL . . . . .	156
INFLUENZA. BY J. FOSTER PALMER, ESQ. . . . .	156
ORGANISATION FOR THE COLLECTIVE INVESTIGATION OF DISEASE . . . . .	160
MEMORANDA AND CARDS ALREADY ISSUED . . . . .	169

THE  
COLLECTIVE INVESTIGATION  
RECORD.

---

HISTORY OF THE MOVEMENT.

BY PROFESSOR HUMPHRY.

IN no other calling in life are the experiences of the several members so great as in the medical profession. In no other, that is to say, are the facts which come under notice so numerous and their combinations so varied and so complex. In no other also is the correct observation of the facts so difficult and the generalization from them liable to so much error. It is an almost necessary sequence that in no other calling is there so much waste of experience, so many facts that pass unobserved, so many that are incorrectly observed, and so many that are wrongly reasoned upon—no calling in which there is so much need for a cultivation of the habit of careful observation in the first place; of the collation, upon a large scale, of well-observed facts, in the second place; and, thirdly, of mature deliberation, and, if possible, wise generalization upon the facts so collated. Hitherto the work of this kind has been done almost exclusively by individuals who, having special opportunities and perhaps special gifts—that of industry being the foremost—have observed, collated their own observations, reasoned and dictated upon them. The advantages of this method are obvious; and it must continue a great means of advancing medical science. But it has the disadvantage of leading to a sort of imperialism or Cæsarism, and hence to the domination, at particular periods, of the dicta or dogmas of

certain persons, and that prevalence of fashion, that blind adoption of shifting views and modes of treatment, which tends so much to repress thought and stifle inquiry.

The collection of information upon a grand scale, which has the double effect of stimulating many investigators and of drawing facts from many sources, not as a substitute for, but as an addition to, individual research, has long been felt to be one means of meeting the difficulties and disadvantages just mentioned; and it has occasionally been attempted on a small scale, in a more or less spasmodic manner, and usually with the result of disappointing those who took part in it. It was obvious, however, that the work ought to be done; and it was also obvious that the British Medical Association, with its enormous force of numbers, its JOURNAL, its organization of branches, its funds gathered by good management and economy, afforded an opportunity, such as had never before existed, of doing it.

Both Dr. Arthur Ransome and Dr. Mahomed urged this upon the Association in letters which appeared in the JOURNAL (Oct. 8, 1864, and Jan. 3 and 10, 1880);\* and feeling that the Cambridge meeting in 1880 was a fitting opportunity for bringing the subject forward, I ventured to do so in my presidential address (JOURNAL, Aug. 14, 1880, p. 244), pointing out the undeveloped capacities for good collective work which the Association possessed, and the advantages to the Association, the profession, and the public, which would accrue from the successful utilization of those capacities. I felt that if the members could combine for social and political purposes, they ought certainly not to hesitate to do so for the promotion of the science and practice of medicine. It was a source of great gratification to me and to all who had been anxious for such a movement, and at the same time it was an earnest of success, that immediately, on the conclusion of my address, Mr. Husband, who is so deservedly respected for his long and great services to the Association, gave the first practical impulse. After saying that "no more valuable suggestion had come before the Association, and it was to be hoped that this one would, like seed upon good ground, be cultivated and bring forth

\* Dr. Pearse, of Haslemere, in a letter to the JOURNAL of Jan. 19, 1881, foreshadowed, as possible, much such an organization as is now at work.

good fruit," and that "the Association had now, he was rejoiced to say, the means—the intellectual means and the pecuniary means as well—of doing that which was necessary for the welfare of the Association," he moved, and Mr. Lister seconded, a resolution, which was unanimously carried, "That the thanks of this meeting are due to the President for his valuable suggestions respecting collective action in accumulating the data of medical knowledge, and that the Committee of Council be requested to consider how such suggestions can best be carried into effect." The Committee of Council lost no time in appointing a "Sub-committee on Collective Investigation," consisting of Mr. Husband, the President of Council (Dr. Carpenter), Dr. Sieveking, Dr. B. Foster, Dr. Allbutt, Dr. A. Ransome, and Dr. Mahomed, with myself; and at the annual meeting at Ryde the following Report was presented and unanimously adopted (JOURNAL, Aug. 13, 1881, p. 297):—

In presenting their report, the members of this Committee are aware that though the contemplated work is one of much importance, which may be productive of very valuable results—one which is worthy of the British Medical Association, and which ought to be undertaken by it—yet it is one which will require great and continuous effort to carry it on in an efficient and satisfactory manner. To combine a number of men in the systematic and careful observation and record of facts is difficult under any circumstances, and especially so in the case of medical men whose irregular and harassing avocations necessarily disincite them to enter upon and continue a labour of this kind.

It is obvious that success will much depend upon the energy, perseverance, ability, and judgment of the Secretary to the Committee which is proposed. The work that will devolve upon him will be laborious; and though it will no doubt, to some extent, bring its own reward, the Committee feel that the Association could not expect that such a task should be undertaken gratuitously.

The Committee further feel that it may be desirable to make some remuneration to those persons who shall be found to have given the time and attention which is requisite to make careful observations and record them well; and they think it will be agreed that a portion of the funds of the Association can scarcely be better employed than in inducing the individual members of the Association to contribute their share to the advancement of medical science by a careful and systematic observation and record of the facts which come under their notice.

The Committee accordingly have agreed to propose the following resolutions:—

1. That a Committee of Seven be appointed annually at the Michaelmas Quarterly Meeting by the Committee of Council, to arrange, superintend, and direct the work of combined observation, and be named the "Combined Observation Committee." That the Committee have power to add to their number.

2. That the Committee meet at such times and places as they think fit, and report at least once in each year to the Committee of Council; and that their Report be presented at the Annual Meeting of the Association.

3. That the Committee shall have power to nominate for appointment by the



Committee of Council a Secretary, who shall be paid (£200 annually) from the funds of the Association, and who shall act under the direction of the Committee, and shall hold office during their pleasure.

4. That the Secretary shall attend such branch meetings of the Association as may be desirable for the purpose of explaining the nature and objects of the investigations, and of interesting and directing the members of the Association in the work.

5. That the travelling and other necessary expenses of the Secretary, to the amount of not more than £100 in any year, having been submitted to and approved by the "Combined Observation Committee," and the Committee of Council, shall be paid out of the funds of the Association.

6. That communications to the members of the Association, and others, for the purposes of the investigation, shall be made through the JOURNAL, or from the offices of the Association, in accordance with the usual regulations.

7. That the Branches of the Association be invited to appoint Registrars who may assist in the work, and that such Registrars shall, together with the "Combined Observation Committee," form a "General Committee" to determine from time to time the subjects for investigation, and the manner in which such investigations shall be conducted.

The following have been suggested as likely to form suitable subjects for combined observation. They are merely mentioned to indicate the kind of work which is contemplated. It would rest with the General Committee to consider their suitability, or to select others.

1. Records of the medical life-history of patients, including the sequelæ of various diseases.
2. Records of the relationship of certain specified diseases;—as, cancer, tubercle, syphilitic degeneration, osteoarthritis, chorea, &c., to any other diseases.
3. Observations respecting epidemic diseases in given districts.
4. The incubation period of contagious diseases; and the duration of contagion.
5. The origin of contagious diseases.
6. The collection of evidence as to the effects of certain remedies.
7. The geographical distribution of diseases.
8. Anthropometrical observations, especially in relation to disease.
9. The hereditary influence of race, climate, occupation, food, &c., in the production of diathesis, or of tendencies to certain diseases.

(Signed)

G. M. HUMPHRY.

*Chairman of the Committee appointed to consider the  
Question of Collective Investigation.*

The Committee formed in consequence of the adoption of that Report, and which has always been called the "Collective Investigation Committee," consisted in the first instance of the members who had formed the sub-committee of the Committee of Council. Dr. Mahomed at first acted as Honorary Secretary, but was, after a time, induced to receive the salary assigned to the office of Secretary; and it is to his ability and unceasing energy that so



many persons have been induced to assist, and so much good result has been attained. The various branches of the Association responded to his appeals to form committees; eminent men undertook to draw up memoranda, to assist in framing cards of inquiry, and to analyse and report upon the results of the information obtained. If, on the one hand, it be felt that the expenses are considerable, it must, on the other hand, be remembered that rarely has so much time and labour been freely given by various hard-working men in the hope that the members of the profession would appreciate and respond to their efforts. It was no small encouragement when Sir William Gull, Sir James Paget, Professor Acland, and others, gave their weighty sanction and approval at the Jermyn Street meeting last autumn; but the greatest encouragement of all has been the number of carefully filled cards which have been received, especially on pneumonia, from the members of the Association living in the various districts of the country. For this shows, not only a widely extended perception of the need of the work, but the willingness of a large number to assist in carrying it out.

It was soon found that the amount of material accumulating was such that it would be necessary to relieve the *JOURNAL* of much of it, and that it was of such value as to deserve a separate publication, which would have the further great advantage of presenting the information collected in a compact form convenient for study and reference. It was therefore agreed at the meeting of the Committee of Council in April, that an 8vo. volume, under the title of the "Collective Investigation Record," should be issued early in July. The additional material which has since accumulated has not only shown the necessity of this, but the necessity also of further issues of the same kind; and, unquestionably, nothing would contribute so much to the success of the movement, to inspire confidence in it and to promote and give value to the results obtained from it as the publication, at stated periods, of the memoranda, results, original contributions, and other documents connected with the work of Collective Investigation.

This volume is therefore offered to the members of the Association and other members of the profession, in the hope that it will prove worthy of their acceptance, and that it may be the

first of a series which, by leading to the larger collation and better consideration of carefully observed facts, will exert a growing and beneficial influence upon the science of medicine.\*

The Committee have been fully sensible of the difficulty and responsibility as well as the importance of the task assigned to them, and also of the failures which have attended some efforts in the same direction; but they feel that difficulty and responsibility are the proper stimuli to exertion, and that failures are often the necessary preludes to success; and they feel that no higher objects of ambition could be entertained by a Medical Association than those of infusing among its members the spirit of scientific observation and of combining them in the work of advancing the theory and practice of medicine.

\* The Committee already have in hand sufficient material to fill more than another such volume as the present, and day by day the returns are rapidly accumulating.

## AN ADDRESS ON THE COLLECTIVE INVESTIGATION OF DISEASE.\*

BY SIR WILLIAM W. GULL, BART., M.D., F.R.C.P., F.R.S.,

*Physician-Extraordinary to Her Majesty the Queen.*

MR. PRESIDENT AND GENTLEMEN,—When invited by you and the Council of the Metropolitan Branch of the British Medical Association, to address this meeting on the Collective Investigation of Disease, I gladly accepted the honour, since the whole sympathies of my life are in the direction of this movement.

After the admirable addresses made in different parts of the country on this subject, first at Chester by the late Dr. Hughes Bennett, Dr. Waters, and Dr. Ransome, then at Cambridge by Professor Humphry and Dr. Mahomed, and lately at Birmingham by Dr. Foster, Mr. Macnamara, Professor Haycraft, and others; though I cannot hope to present this matter in any new aspect, or to add anything to the arguments in favour of it, still I am glad of an opportunity, in conjunction with Sir James Paget and others on this occasion, to take part in promoting what promises to be of so much advantage both to the public and the profession.

This meeting may be considered as a supplement, though it is but accidentally so, to the Bradshawe Lecture, given a month ago at the Royal College of Surgeons by Sir James Paget, in which he endeavoured to draw the mind of the profession into new lines of pathological inquiry, and to consolidate the results by the formation and extension of museums of morbid anatomy.

\* Read before the Metropolitan Counties Branch of the British Medical Association.

Now, although morbid anatomy is at an immeasurable distance from a great part of medical pathology, as physiology is a distinct science from anatomy, still both are built on anatomy. Without morbid anatomy our work would be foundationless and in the air. Nevertheless, my object to-night is to direct your attention to, and to enlist your services in, the investigation of a region of facts, which in large part, at least, lies at a far distance from the gross mechanical terminations of disease as they come before us in the *post-mortem* room. And although the hoped-for gains of this collective investigation movement may not be such as to admit of being labelled and placed on the shelves of a museum, they will serve to throw a new light on what is already placed there, and will at least help us to a better knowledge and practice of our profession.

You will admit that a migraine headache, an attack of asthma from the smell or sight of some particular object, the troubles of digestion from mental work and anxiety, the special liabilities to disease of certain families, and the cloud of small ailments which often make life intolerable, though having no morbid anatomy, deserve a more exact study than they have yet obtained, whether for the satisfaction of the intellect, or for the latent pathological meaning they may convey to us; and especially, further, that when we have learned to question Nature, we find she has much more to tell us, even on trifles, than we had expected.

The idea of a collective investigation of diseases, in which every member of the medical body shall have his effective part, though obviously not to be realised at once—not, indeed, until the sun of science, which is the true Apollo of Medicine, has risen far higher in our sky than now—is yet one that at once challenges our ready and best efforts for its realisation.

Whilst the morbid anatomist is engaged in our hospitals and medical schools in demonstrating the effects of disease on the several organs and tissues of the body, we desire that all the practising members of the profession over the country, in the colonies, and in other parts of the world, should assist in the inquiry as to the origin of diseases—their early symptoms; their mode of spreading in families; their combinations; the causes of their intensity; their modifications in individuals; in families; their occurrence according to time of year; locality; sanitary condi



tions; occupations; and many other circumstances, some as yet but dimly discerned, and others not yet suspected. The value of this movement will, I believe, be obvious to all after but little consideration, for it will be admitted that had we leisure, proper means at our disposal, and from previous training a fitness for exact observation, we should find in general practice one of the most valuable fields of pathology, as here and here only we have before us the earliest signs of departure from health, and the only opportunities for tracing the course of a disease from its beginning to its end. Having passed many years in hospital and private practice, I have come to see that experience gained in the latter is necessary for the correction of that acquired in the former, especially as helping towards a truer pathology.

In hospitals we have more largely to do with organic lesions and with isolated cases of acute inflammations or developed fevers, and in all with an incomplete personal history and without any family record. We cannot thus learn with any exactness either the beginnings or endings of disease. Patients come under observation with their maladies far advanced, and they often pass from observation but imperfectly cured, thus leaving fallacious histories, both in pathology and therapeutics; and if they die, morbid anatomy can often give but a confused and inextricable mass of facts, which it may be difficult or impossible to put into their true relations. One might as well hope to determine the physical geography of a country, by measuring and analysing the contents of its rivers as they fall into the sea, as to hope to reach a true pathology from studying alone the results of disease on the *post-mortem* table. Let it, however, be remembered, that we still insist upon the fundamental necessity of morbid anatomy as the only basis of true advancement. I am glad, therefore, to be informed by Dr. Mahomed that it is in contemplation to associate paid pathological experts and morbid anatomists with this movement. By such means alone can we make our results permanent stepping-stones for those who follow us.

As a passing illustration of what is here insisted upon respecting the relation of pathology to morbid anatomy, and as indicating what we may expect from wider research, pardon me if for a moment I refer to renal diseases. One of our recent and best writers on the subject concludes by expressing his conviction that



“there is but one Bright’s disease;”—this honoured name of Bright defining a state found after death, and fixed upon as the battle-ground of renal pathology—and I see that the latest statement of to-day, drawn from experiments on animals, is to the effect that destructive changes in the kidneys have but one form and lead to but one result in the tissues of these organs.

I appeal, however, with some confidence to our present limited experience in general practice, and with more to that wider experience which will be gained by Collective Investigation, whether there are not to be found quite different beginnings, quite different courses, and conditions requiring quite different therapeutics, for that which in the *post-mortem* room is regarded but as one pathological state.

In disease, one stream of morbid action naturally falls into another; and, whilst morbid anatomy gives us the final synthesis of results, there is but one possible means of analysis, and that through noting beginnings, order, and progress. Diseases are apt to so far assimilate as to become much alike towards death; but our work lies in a far other direction.

Believing, as I do, that more than we dare now expect will grow out of Collective Investigation, we must not be over sanguine as to its immediate fruits. The essentials for success are not only the *numbers* but the *intellectual organization* of the movement. If we aspire to have the arms of Briareus, we shall need the eyes of Argus; for Nature is very much a Sphinx, and will answer no question put to her if it be open to evasion. Yet in truth it is not so; for truth lies on the surface, if we had minds trained, and free from prejudice, to see it.

The plan of this movement at present is to draw up memoranda on the several subjects for inquiry, and to issue with these cards of questions to be answered. Already such important memoranda have been issued on Acute Rheumatism by Drs. Goodhart and Barlow; on Acute Pneumonia by Drs. Sturges and Coupland; on Inherited and Acquired Syphilis by Mr. Macnamara and Dr. Barlow; on Diphtheria by Mr. Shirley Murphy; and questions on the evidence of the Contagion of Phthisis by Dr. Burney Yeo. This plan leaves nothing to be desired.

Nothing can be more useful and instructive than such memoranda. When extended over the whole range of medicine, they

will place before the practising members of the profession in every locality, more or less succinctly, the state of our knowledge on the different subjects proposed for inquiry; and they will, whilst they indicate what we want to know, inform us of our ignorance on the various subjects.

The main difficulty lies in properly formulating the questions to accompany the memoranda. They must of necessity be so simple, pointed, and incisive, as to admit of no vague answers. This, without saying it, calls for a great amount of knowledge and intellectual combination, and no slight mental perception; for rightly to ask questions of Nature is the highest science of the intellect.

This is, indeed, the vital centre of the whole movement. If the Committees issue for any inquiry a definite question, and, that being settled, follow it up by another, and so on in series, Nature must at last be driven into a corner, and be obliged to say "Yes" or "No."

If it be that truth is hidden in Nature as a stimulus to the intellect in the general pursuit of knowledge, to us this obscurity of things has a double meaning where duty and interest come in to urge forward the pursuit.

And here we ought to remind ourselves that, if there is an experience which teaches, there is a much larger experience which is fallacious. Hippocrates rightly begins his aphorisms with this reminder.

Numbers, without perfect organization, can effect but little or nothing. We require not only the fulcra of ascertained facts upon which to base our movement, but such intellectual combinations and direction of effort as is in a manner required in mechanics. If, says Bacon, men had attempted mechanical labours with their hands alone, and without the power and aid of instruments, as they have not hesitated to carry on the labours of their understanding with the unaided efforts of their mind, they would have been able to move and overcome but little, though they had exerted their utmost and united powers. Yet men, he adds, are hurried on by senseless energy and useless combinations in intellectual matters, as long as they expect great results either from the number and agreement, or the excellence and acuteness of their wits.

And we may add, that as great mechanical results cannot be obtained without engines framed by the wit and hands of many men, neither can a knowledge of such facts as we have to deal with be attained without an exact *mental* and *numerical* combination of the members of our profession.

It will perhaps, and naturally, be objected that it is almost impossible to organize for any useful purpose the labours of men already overburdened by the cares and fatigue of practice, and that there is neither time nor fitness for delicate inquiries on their part. Admitting that this objection is valid, it may be urged in reply that it need not be insuperable; that if this movement makes some demand upon the busy practitioner, he will (as remarked by Mr. Macnamara in his speech on this subject at Birmingham), in proportion to the help he affords in carrying on this work successfully, receive back quite as much as he gives. That, further, it cannot be denied that when we see the meaning of the apparent trifles which in practice would otherwise oppress and worry us, our burden is thereby much lightened, and that nothing could encourage us more than to feel that even one daily observation recorded was adding to our general store of knowledge, and making the path of practice more easy. There is no tonic to the mind greater than the sense of work done; and our journey is likely to be made shorter, as it certainly will be easier, if the way is illuminated.

We, indeed, owe it to those members of our profession, who are admittedly overwhelmed by the apparently senseless details of their work, to promote a movement like this, the object of which is to bring order into their chaos, and to help them to stamp a scientific value upon facts hitherto only burdensome. If we compare the unflagging interest of any pursuit where the aim is high and clear with the tediousness and wearisomeness felt when working in the dark, we shall readily admit that we are actually lightening the burdens of practice by thus adding to them, and by giving some portion of them a sense and meaning.

It is the spirit of a man which enables him to do his work lightly and cheerfully, and he will certainly be helped in this by a combination with fellow-workers on the same subject.

There is, however, a further consideration which should weigh with the practising members of our profession. This combination



for the purpose of extending medical knowledge is an important duty in itself to all concerned, for however slowly, and at first imperfectly, such a combination may operate, the smallest progress is a great gain to society and to our profession, and if favoured as it plainly claims to be by all the members of it, and helped by the efforts of our best minds, cannot but, in the course of time, lead on from what is now but dawn to daylight.

As to the objection on the score of fitness, if this were urged as an argument against this movement, it would stand self-condemned on every ground; for if the members of our profession are unfit to observe, they are largely unfit to practise.

Probably not so much as this is meant, when it is said that the working members of the profession cannot help us much, but rather that until the special workers in the sciences of physiology and pathology have made more advance, it would be useless for untrained practitioners to attempt anything novel.

But we believe this to be one of the fallacies of the day, and one of the causes which retard the progress of practical medicine.

Without in any degree depreciating the more recondite study of physiology and pathology, whilst indeed hailing with thankfulness the light such studies give us, and honouring those who in that behalf work for us, we may fairly maintain that we have been too indifferent to the value of the facts which lie nearest to us in our daily work. The feeling that only experts in science can do anything, and that we must wait until they, from their centre, move us, or we shall not move successfully, is only partially true.

To say that this Association is beginning at the wrong end, is to misapprehend how knowledge is gained. The history of medicine is in our favour. We do not wait to discuss the nature of sensation; or how it is that the peritoneum is painful in inflammation, before we give opium for its relief. Currie proved the use of cold effusion in fever more than half a century before physiologists began to suspect that there was a nerve-centre in the cord for controlling animal heat; and as yet they have not taught us specially how to utilise their discovery. We have long known the curative effects of quinine in ague, though physiologists have not yet determined the operation of the *miasm-organisms* which are present, and which may give rise to this disease. Laënnec

developed his method of physical examination of the chest on the commonest and nearest principles.

All practice obviously lies in a knowledge of proximate facts, and it is equally obvious that that practice will be most guarded and exact which is guided by a knowledge of all the facts, both near and remote; still we are not to esteem lightly that which is near, because we are ignorant of that which is remote. It is good knowledge to have learned that fire burns and water wets; though obviously it is much better knowledge to know all the facts of combustion and wetting; the combinations, the oxidations, the adhesions, the capillary attractions, the amalgamations, the endosmoses, the exosmoses, and the many other molecular changes which attend these processes.

It would appear to be one of the faults of the medical education of to-day, which this movement may in some degree correct, to lay undue weight upon ultimate facts, whilst we neglect those which are near; to indoctrinate the student with the belief, for instance, that if he can run off upon his fingers the supposed ultimate constitution of the gastric juice (about which we are still very ignorant), that he has learned something respecting the digestive process; or that, by reciting the atomic composition of an organic substance, as muscle, he has learned something about it, though, in fact, nothing but what appertains equally to mere dead substance.

It will not be the least valuable result of this movement, if it correct our prejudices in these matters; and hence it has already been well observed that one of its effects will be educational on the whole profession, from the youngest student to the oldest practitioner. It will quicken and keep alive a sense of docility, the want of which is the cause of that confirmed prejudice which we often call experience, and which blocks the way of progress.

If the eye sees no more than it brings with it the power to see, then any objection against fitness must be met by the several Committees of the Association, when they issue their questions, by accompanying them with such memoranda as instruct the working members in the way of observation in any particular direction.

We, of all men, ought to be the last to be discouraged by the small promise of beginnings. Had we been present at the dawn of



organic life, we should hardly have predicted its wonderful results as seen in past and present times. This collective investigation has been started on the principle of giving significance to what has hitherto been neglected; it would therefore be directly contrary to the *animus* of the movement if we underrated the power and probable success of efforts which may at first produce but little fruit.

The first intention of the Association respecting this movement was to obtain a better notification and more complete statistics of disease; and this was carried out to some extent by Dr. Ransome in Lancashire and Cheshire, but apparently not much came of it. The present form of the movement is chiefly due to Dr. Mahomed and to the advocacy of Professor Humphry at Cambridge in 1880, and subsequently to the meeting lately held at Birmingham.

Fifty-four Committees have been already organized, one of the Branches having six Committees, and another four. These Committees include from eight hundred to one thousand of the chief practitioners in England, Scotland, and Ireland, already pledged to the work, Dr. Mahomed acting as honorary secretary to the whole, and who may be called its moving spirit.

It is not my province on this occasion to indicate specially what should be the subjects for inquiry, or to discuss the memoranda which should be issued upon them, but I may, perhaps, be permitted to refer to the subject generally.

For instance, on the contagiousness of phthisis a preliminary difficulty occurs as to what is meant by phthisis. Does the term include all the cases of destructive pneumonic changes, beginning from the apex, or only those of a distinctly tubercular type?—cases beginning acutely with fever and hæmoptysis, and ending fatally in a few weeks, and others with pleurisy or bronchitis, and lasting perhaps twenty years,—or only those springing up without much observation until the infiltration of the lung is far advanced? To properly distinguish the cases will be an affair of much difficulty, since they approach from various points and intermingle inextricably. If the evidence of the communicability of phthisis should appear to gain force by extended inquiry, it will then obviously be necessary to determine with more than common accuracy in which form of phthisis this communicability occurs. If only in the more chronic forms, then

whether the phthisis of old age is to be classified in this respect with the phthisis of the young.

It is plain that if in this movement of collective research we see a land of promise before us, we must at the same time recognize that the way to it is by a narrow and dangerous path. For whilst truth must be accepted when it is proved to be such, nothing will be more pernicious than false conclusions and partial truth sanctioned by so large a body as this. The opinions of a single individual would have less weight, and would take longer to permeate a profession, than conclusions having the colour of authority, and where the error would be perpetuated by numbers interested in the fallacy.

Assuming, again, respecting this question of the communicability of phthisis, that the presence of bacteria in the expectoration is characteristic of the disease at a given stage, we should then have to determine whether they were present at all stages; and whether the phthisis bacterium received its specific form from the exudation in which it was found, or whether its presence determined the specific form of the exudation. The fallacy of putting effect for cause is here imminent; and families might be broken up and society much alarmed by conclusions having but little foundation.

As genealogical trees, showing the rise and extension of families, with their many collateral branches and intermarriages, are found in great houses, and are regarded with pride and veneration, so we could wish that, in a like manner, *life-histories* were found in every family, showing the health and diseases of its different members. We might thus in time come to find evidence of pathological connections and morbid liabilities not now suspected; and we might discover means of prevention by a better knowledge of the origin and extension of maladies through blood-relation. The proposal of constructing such family life-histories is an important part of this scheme of Collective Research; and more would certainly be gained socially by the genealogies of health and disease, their connections and causes, and by tracing the strength of the strong as well as the ailments of the weak in a family, than from such barren histories as I have referred to, which tell only of inherited fame, but do not indicate the way to perpetuate and augment the inheritance.

Unfortunately, there is a feeling of safety in ignorance; and there prevails in us a sort of blind superstition, a survival of the darkest ages of man, which makes us think that there is a kind of religious trust in not seeking too minutely into the ways of life; as if the intellect of man were the servant of impiety rather than as it is—the handmaid of all that is good to us. Again, there is another hindrance to our obtaining all we want in this matter of family history: “all men think all men mortal but themselves,” and there is a half-conscious sense of shame in admitting any liability to human frailties. This makes it a great difficulty to obtain the truth we want, though we may well excuse it, and believe it to be an instinctive tribute to the righteousness of Nature’s laws; and an unconfessed confession, that many of our diseases and weaknesses are due to our own fault, and ought to have been prevented, as, we trust, they will be through this movement in the course of time.

This matter of life-histories is no new subject, though it is one to which this Association especially wishes to give a new impetus. All practitioners of large experience would be able to tell us something concerning the associated occurrence of diseases in families, which are not generally supposed to have any connection between them.

Dr. Cheyne, in his article on epilepsy, in the *Cyclopædia of Practical Medicine*, says we conceive that epilepsy is as certain a manifestation of the strumous diathesis, as tubercular consumption, psoas abscess, etc. Now it is, of course, not to be maintained that tubercular disease and epilepsy are one state; but it does not seem improbable that diseases from hereditary defects of organisation may evince themselves in most different ways, and that there may be a common underlying bond of pathology between them. No advance of our knowledge on these and many similar points can be made through morbid anatomy. It is only through family life-histories that the subject can be studied. Such histories, incomplete as they now are, are often of great use in practice. Take, for instance, the anæmia and languor so much complained of in the girls of a family. Though, of course, such ailments are often due to the want of physical and intellectual training, they are by no means altogether so; and certainly the most difficult and intractable of them are not to be so explained.



We must, in many instances, have the life-histories of the parents or more remote ancestors, before we can fully unravel the causes of irregular menstruation, hysteria, anorexia, uterine fluxions, and the like. And the same line of inquiry applies to the headache, pallor, dyspepsia, and seminal hypochondriasis of the males of such families. Further, take another example in acute rheumatism, upon which already a memorandum and questions are before the Association. The hereditary transmission of the rheumatic diathesis, its occurrence in intra-uterine life (as appears to be shown by some of the congenital cardiac malformations), and its association with other diseases prevailing in the same family, throw a light on its pathology not to be gained at the bedside, or in the *post-mortem* room.

Life-histories, as Sir James Paget pointed out in his Bradshawe Lecture, would give us the genesis of new and rare forms of lesion, and I hope I may add they would also show how the organic laws, favoured through generations, prevail over and wear out disease from the stock.

Again, if I may be allowed to refer once more to the still vexed question of the pathology of the contracted kidney and its relation to cardio-vascular changes, my friend Dr. Sutton and myself believe that it is only by a careful record of the life-histories of such cases that the matter can be settled. We believe that it will then appear that the same pathological tendency in the arterioles may give rise to lesions in various parts, and not in the kidneys only; to the retinal apoplexy in one, a cerebral apoplexy in another, to chronic changes in the cord or brain and cord in a third, to contraction of the kidneys in a fourth, and in all to muscular changes in the heart.

Family life-histories would show whether it be true or not that the ailments of childhood and age—eczema, bronchitis, diarrhoea, etc.—have a parallelism in their pathology. Certain it is, they have much in common as to their occurrence. In old age, these ailments are called gouty, and in infancy and childhood catarrhal. It would be a point gained, if it were cleared up, whether, in fact, childhood is as gouty as age; or whether the word gouty, as applied to the diseases of the latter period, has any actual pathological value. If an old man's urine deposits largely crystals of uric acid, whether he be distinctly gouty or not, we refer it

to that state; though such deposits are as frequent in infancy and childhood; and uric acid is the chief component in the urine of many creatures lower than man. Varied and numerous family life-histories might place on a surer basis our knowledge in this and a thousand matters.

Take the inquiries of to-day respecting infectious diseases, and the increasing evidence that such states are due to agents which we may hope to fix and analyse. These, if ever completed, will still leave untouched a vital question in the solution of which this collective research will have to take an important if not an isolated part;—I mean, how it is that the same poison acts with such varying intensity in different families; in some, the organism breaking down at almost the first touching of the poison; others suffering but little, and others having almost or altogether immunity. Does this depend upon certain family peculiarities, and if so, what? Does the immunity come from ancestors having passed through the ordeal, as occurs to the individual in vaccination and syphilisation? Family histories will show us how far these immunities and susceptibilities extend; and with what peculiarities of the nervous system they are associated, and how acquired. Allied to this inquiry, is also that of the action of endemic poisons upon the new-comers into a district; at which I can only glance.

In the early part of this address I asserted, that Collective Research would give a new colour and meaning to many ailments, and show their relations to later organic changes; and I will conclude what I have to say, by referring to a set of ailments which very largely claims the attention of the practitioner, and a fuller investigation; I mean a set of cases whose life is nothing but ailment. Their physiology, if I may so express it, is a state of pathology. Their nervous systems are so sensitive, and their digestions so feeble, that they know nothing of that satisfactory resistance to disturbing causes from without, which we call health; and who often suffer as much from futile attempts to cure them, as from their congenital defects.

These cases supply a constant *clientèle*, and I can hardly say how much we should add to the happiness of mankind by a better understanding of them. Of course, I exclude those dilettanti *malades imaginaires* who may be said to enjoy bad health; whilst



the class of cases to which I refer of right claim our greatest sympathy.

But I feel that I must not longer tax your patience. From the nature of the case, it would be impossible to indicate, even in a cursory way, the many bearings which this Collective Research may take. One thing is certain : every question settled will but open another, and give renewed encouragement for fresh exertions. In all its aspects this project, set on foot by the British Medical Association, cannot but advance the highest interests of our profession, and the more surely, in proportion as it extends our knowledge of the prevention and cure of disease.

This is the age of combinations ; but I know of none which has a purer object than this ; for what can surpass that whose purpose is the investigation of truth for the good of man ?

## AN ADDRESS ON THE COLLECTIVE INVESTIGATION OF DISEASE.\*

BY SIR JAMES PAGET, BART., F.R.C.S., F.R.S.

*Sergeant-Surgeon to H.M. the Queen.*

MR. PRESIDENT AND GENTLEMEN,—When it was proposed to myself, as well as to Sir William Gull, that we should give addresses here on the subject of Collective Research, it seemed to me scarcely possible that two addresses on the same subject could be given on the same night; and when Sir William Gull was good enough to promise that he would speak first, I felt nearly certain that he would leave me nothing that could be said, except after the manner of useless repetition. I shall, therefore, speak very briefly; and I will give emphasis in proportion to the shortness of my speech, by declaring that I entirely agree with everything that Sir William Gull has said. I will ask you to allow me to flatter myself so far as to say that, if I had spoken first, I should have said the very same things. If I may condense what Sir William Gull has said at full length, it seems to me he has shown perfectly how this design of the British Medical Association may fulfil the two great purposes which we should have constantly in mind in our profession—the increase of knowledge and the improvement of the mind of the observer; for these things can rarely go apart.

I cannot but admire how well a large assembly such as this, representing, as it does, a yet larger Association, has declared itself to be in the state of mind most favourable to the acquirement of knowledge—the state of partial ignorance. The list of questions which is published upon each of the papers sent out by the Committee indicates that we are not ashamed to confess our

\* Delivered before the Metropolitan Counties Branch of the British Medical Association.

doubts on some of the most important things that come before us ; that we are prepared to start confessedly ignorant on many points upon which we are supposed to have complete and final knowledge. I think that, in common with most scientific men, we may hold that this, though rare, is a just state of mind, even though there are large groups of men, and those much esteemed, who rarely express doubt on anything, and thereby command the assent of those who listen to them. Without expressing the smallest preference for one side more than the other, I would say that this is especially to be found amongst politicians, in whose speeches we almost entirely miss the words which are most familiar to ourselves—"perhaps," "possibly," "I rather think," "I would venture to suggest." I have looked with much curiosity, not for the sake of acquiring political knowledge, but for the sake of comparing the political and the scientific mind, to see if in some of the best and most renowned speeches I could find one expression of the kind. Not one is there. We must therefore be content to put up with what may be regarded as a sort of unpopularity if we confess ourselves to be always beginning with doubts, in order that we may be more sure in proceeding towards knowledge. Certainly there is no state of ignorance so hopeless, so profound, as that which cannot even doubt.

The questions also show well how very large the inquiry is, and how various the objects that must be had in view in every research that we undertake. Sir William Gull has rightly said that I had in view in the Bradshawe Lecture the promotion of museums of morbid anatomy, and I am quite conscious that, whenever one undertakes to promote one thing, it is very difficult to avoid an appearance of depreciating others. I put, as it were in parenthesis into the lecture, some words which probably attracted no attention, and might have been omitted, implying that I should be the last to hold that any one method of inquiry on any scientific subject can be sufficient ; and I said, what I venture to repeat, that neither the pathologist who thinks lightly of observation in practice, nor the practitioner who thinks lightly of observation in pathology, will attain to more than that partial view of truth which is nearly as bad as error. And so I think, although still very highly estimating pathology as illustrated by morbid anatomy, that I may nevertheless speak with an unbounded desire for the

success of an inquiry such as this, in which morbid anatomy may be almost wholly left out of sight. It is impossible to reckon what shall be ultimately the relative degrees of importance of the several methods of inquiry. I would rather hold that it is altogether childish to endeavour to say which method is best. One rule may be held for all; that is for each man best which he can do best, whether by force of circumstances, or by his own natural ability. And as one cannot but observe that the chief design of this collective inquiry is to bring into the field of knowledge all that may be gathered in general practice by family and general practitioners, so I would not hesitate to name the knowledge which may be so gained as probably likely to lead to the very highest knowledge, and even the solution, of the most difficult problems in pathology that can be set before us.

I would have dilated on this subject, but that Sir William Gull has treated it so fully in regard to the attainment of family histories. There is certainly no other means so good as that which may be possessed by those who have known families for generations, and who can, of their own knowledge, and not on the fallacious and often very false reports of relatives and friends, declare what has been prevalent in this and that household. And yet, if one should set before one's self the gravest and most important problem in all pathology, it would be that which concerns the inheritance of disease; and, as Sir William Gull has rightly stated, the inheritance, not of disease alone, but of that which from generation to generation shall gradually obliterate the disease which one ancestor may have acquired. And let me observe that this is a kind of knowledge which can be gathered in the most ordinary pursuits of life; it needs no minute inquiry. The mere recollection of the daily life that has been spent in this or that small village, among the two or three generations of one family, may recall it at once; and this is what we most thoroughly need, this personal and exact knowledge. When I speak of knowledge that may be gained in the study of common things, there always comes to my mind the great example of Darwin, whose renown will last as long for the manner in which he pursued his knowledge, as even for the grand knowledge which he acquired. I have often felt in Darwin's greatest inquiries, and I would cite, as the chief among the kind, his last work on the



influence of worms, that there are very few facts which might not have been observed by the common daily labourer in the field. He had the rare power of taking the common things that other men waste, and out of them making the grandest material of scientific work. It is vain to say, in any branch of practice, "I have no opportunity for scientific inquiry; I cannot investigate this; I can contribute nothing to that which I see the scientific members of the profession are doing." It requires merely the opportunity of a practice in the country, and the mind and resolution of Darwin, to bring great pathological conclusions out of the most ordinary facts of daily life in general practice. And if one wanted another motive for this, it would be the improvement of the observer's mind, and the charm which, as Sir William Gull has well said, he would find in the promotion of his own work, and in the mere pleasure of observing and finding his conclusions.

We are all very apt to think that, as we grow older, we acquire experience and grow wiser. I have lived long enough to feel and discern the exceeding fallacy of this as a general rule; and yet I venture to say that the starting with a distinctly observing and scientific mind is of the first importance in our lives; for the whole career of a man's life, his whole real success in practice—or, to speak more to the subject of the evening, the whole value of what he may contribute to knowledge may depend upon whether he begins with and always cultivates a mind for scientific inquiry. I am quite ready to believe, or rather I am quite willing to hope, that the young men who now enter upon life, and are called qualified practitioners, are really qualified, in the broadest and largest sense, as well as in the legal one. Legally they all are, but some are certainly not qualified to make full use of the knowledge which they may meet with in their career of practice. A very wise old man said that, it would be well if the youngest amongst us would remember that he is not infallible. It is a fault which is apt to prevail, to think that at the conclusion of study and with the right to practise, there is full power to do all that is required. There is not. Dr. Billings said cleverly the other day that he wished, after thirty years of practice, he really knew half as much as he was convinced he knew when he first obtained his diploma. I recollect Sir Benjamin Brodie telling me that, when he looked back over forty years of practice, he was



astonished, chiefly, at the ignorance with which he began ; and I think to most of us who have had a long career, the same reflection may often happen. But both of them would have said that the knowledge which they really acquired late in life was due, not to the mere fact of seeing many cases nor to having lived long to see them, but to the care, the prudence, the discretion, with which they observed, and remembered, or recorded what they saw.

If I may impute a fault to those who are admirable in all the ordinary work of their life, I would suggest how large a quantity of knowledge lies scattered and lost to the scientific world in the charge of those who are in large practice, and who record nothing. It will be indeed an admirable result, and I think it will be the result, of this system of Collective Research, if this fault is mended ; if every one can be induced to record his answers to the several questions that will be asked, and in the recording will get the habit of recording for himself many things that are not asked ; for I am quite sure that there is no one who will undertake this but will find in the task an amount of refreshment and of pleasure in his practice, equal to that which may be had in any kind of speculative pursuit, in any kind of sport, in any kind of game. For amongst all these things—in sports, and games, and speculations, and the rest—there underlies one thing—the desire to unveil mysteries. Even the boy who tosses a halfpenny to see which way it will fall may illustrate a part of the scientific mind at every toss he invents a mystery ; at every fall he solves it. So on a larger and grander scale, in every investigation that we enter upon, we set before ourselves a mystery—a mystery that may be as interesting as that of a romance, of a drama, of a great tale told in the history of past times. Mystery is before us ; the power of solving it may be in our minds ; and I venture to promise to all who will begin with this collective inquiry, and then proceed from it to personal inquiry, a pleasure as great as can be had in any of the pleasures of life.

## A REPORT ON THE COMMUNICABILITY OF PHTHISIS.

THE recent authoritative publication and wide diffusion of a doctrine as to the nature and mode of origin of Pulmonary Consumption, at variance with the views previously held on this subject, has excited the greatest possible interest amongst the members of the medical profession in all countries.

Apart from the more strictly pathological inquiries, to which the promulgation of this doctrine and the observations and experiments upon which it has been founded naturally gave rise, it seemed to involve a view as to the probable or possible origin of this disease, by communication from one person to another, which also demanded as complete and as careful an investigation as the circumstances of the case would admit of.

It appeared, therefore, to the Collective Investigation Committee of the British Medical Association to be their duty to take the earliest opportunity of applying the agency of collective investigation to the elucidation of a subject of the highest importance and of the widest and most general interest, not only to the medical profession, but also to the whole community.

It was thought advisable by the Committee that their first effort in this direction should be a very simple one, and that the initiatory inquiries should be such as would admit of immediate and ready answers ; and that it should be left to the observers to answer them in their own fashion. It would then be easily seen whether the subject was one which might be pursued further, and in a more systematic manner, with advantage.

For this purpose, on the 6th of January of the present year the following questions were addressed to every member of the *British*

*Medical Association*, and enclosed in the issue of the *Journal* of that date :—

“Have you observed any case or cases in which pulmonary phthisis appeared to be communicated from one person to another?”

“Please answer ‘Yes’ or ‘No.’”

“If more than one case observed, say how many.”

“In the event of your having observed any case or cases, the Committee will be further obliged if you will give any brief particulars you may think worthy of record. In doing so the following points should be noted :—

“1. The date when the observation was made.

“2. The relationship, if any, between the individuals concerned.

“3. The presence of family predisposition to the disease. It should be particularly noted whether the disease had occurred in the parents, grandparents, and other relatives, or only in the brothers or sisters of the person to whom the disease was communicated.”

In the same number of the *Journal* in which these questions were circulated, an article was addressed to the members of the Association, for the purpose of setting forth the scope and object of the Investigation, as well as to set before them a brief account of the history of the inquiry, so far as seemed necessary for furthering the object in view. The substance of this article was as follows :

“It may be of use,” the article begins by saying, “if we take advantage of the present opportunity, to put before the Association a few considerations on the question whether there be or be not any contagious quality in pulmonary consumption.

“In order that we may be perfectly clear as to the issue before us, it is essential, in the first place, that we should be agreed as to what we are to understand by the word ‘contagiousness’; and it will be advisable for the purposes of this inquiry, that all should regard this term as identical in meaning with the word ‘communicability.’ Any narrower acceptance of the term in the present state of our knowledge would be unphilosophical and prejudicial to the investigation.

“Scarlet fever, typhoid fever, and syphilis, are all communicable diseases. It is universally admitted that they each possess



a special 'contagion'; and it is important to remember that, if there be a contagion of phthisis, it is not necessary that it should resemble either the contagion of scarlet fever, or that of typhoid, or that of syphilis, in any other quality besides that of being under certain conditions communicable from one person to another. The argument that phthisis is not contagious, because it is not conveyed by casual contact with a phthisical patient or with his clothes, as scarlet fever is, would have applied with equal force to typhoid fever and to syphilis. And when it is advanced as a reason against the communicability of phthisis, that in an institution devoted to the reception of cases of this disease, the occurrence of phthisis amongst the officers and attendants has been so rare, that it might fairly be regarded as accidental, it is obvious that this only provides an answer to the question, 'Is phthisis communicable under the conditions that prevail between the patients and the attendants in this particular institution?' and not to the question which now concerns us, viz., 'Is phthisis communicable under *any* conditions? and if so, Under what?' to which would follow the further query, 'Is phthisis communicated by a special contagion? and if so, What is it? and What are its properties?'

"A number of cases have been observed, by trustworthy and careful observers, which have left on their minds the impression that phthisis is, under certain conditions, an infectious disease. What it is desired to know is, are such impressions widely entertained? And if so, what are the circumstances under which they have arisen? The following case, which has lately been communicated by an eminent provincial physician to one of the members of the Committee, may serve as an example of what may be brought forward on one side of the question.

"The remarkable series of cases of apparent communication of phthisis from husband to wife, carefully observed and ably reported by Dr. Hermann Weber in the *Transactions of the Clinical Society*, are now well known to the profession. It is impossible to desire more careful observation and record than we find there. In most of the instances quoted by Dr. H. Weber, the disease, when communicated (assuming, for the sake of argument, that it was communicated), ran a florid course, and was rapidly fatal,

a circumstance which had been observed and noted by Richard Morton two centuries ago. This, also, is a point on which the Committee would be glad to receive evidence, viz., as to the course followed by cases which were supposed to arise from contagion.

"It is well known that, in Italy, it has always been believed that consumption is contagious. In a letter recently received from Florence, the writer states: 'Consumption is considered by all here to be contagious; anyone dying of consumption in a house, the bed is entirely destroyed, and also all the clothes of the deceased, and the room thoroughly cleansed and fumigated, and often shut up and never used.' It is worth while to consider how this belief arose, and why it has been maintained. (Reference to this subject is made in Return 205.)

"Dr. Playter, editor of the *Sanitary Journal*, Toronto, has recently published 'An Etiological Statistical Report' on Phthisis, 'based on the early history of over 250 cases of well-marked tuberculous pulmonary consumption.' And under the head of 'Contagion' he states:—'In about 28 per cent. of the cases, the patients had been more or less with relatives who had been suffering from the disease—attending, nursing, or sleeping with them.' And amongst his deductions occurs the following:—'Consumption is doubtless a contagious disease, though the evidence herein of this is not strong. . . . Besides the exciting cause of a disease, there must be, before the disease can be developed, a predisposing cause—the seed must have favourable conditions for its development and multiplication.'

"Cases of phthisis which have appeared to the observer to have arisen from contagion have been put on record, or referred to by the following:—Richard Morton (1697):—'A contagious principle,' he says, 'also propagates this disease; for, as I have often found by experience, an affected person may poison a bedfellow by a kind of miasm like that of a malignant fever.'

"Heberden (1802) says:—'I have not seen proof enough to say that the breath of a consumptive person is infectious; and yet I have seen too much appearance of it to be sure that it is not, for I have observed several die of consumption in whom infection seemed to be the most probable origin of their illness.'

"Sir Alexander Crichton (1823) had seen many cases in which he believed the disease had been communicated. Dr. Masen



Good (1825) observes: 'I have myself been witness to various cases which could not be ascribed to any other cause' than contagion. Trousseau (1845) expressed a hope that 'the communicability of phthisis may again become at least a matter of dispute.' Copland (1859) stated his belief that 'the disease is caused by infection.' Bowditch, of Boston (1864), after narrating eight cases in his own practice, concludes that it may be 'infectious' under certain circumstances, but not 'contagious in the usual acceptation of the word.' He, however, believes that he has seen it communicated. Dr. Bowditch had issued questions and had received replies from 210 physicians as to the causes of phthisis, and with respect to *contagion* as a cause; 110 answered in the affirmative, 45 in the negative, 27 were doubtful, and 28 did not feel able to answer the question. Dr. Wm. Budd's (1867) article on this subject is well known. Dr. Parkes, alluding to Dr. Bryson's 'Cases in the Mediterranean Fleet,' observes: 'There is some evidence of a pneumonic phthisical disease being contagious.' Many others might be mentioned, as Peterson of Copenhagen, Perrouet of Bordeaux, Alexander Harvey of Aberdeen, Jules Guérin of Paris, Buhl of Munich, Professors Alfred Stillé, J. M. Da Costa (who says, 'I have met with a number of instances which seemed to prove the contagiousness of phthisis'), J. Solis Cohen, &c. On the other hand, we have opinions expressed in opposition to this view by Dr. Cullen (1791), Dr. Benjamin Rush (1809), Dr. T. Young (1815), Laënnec (1818), Sir Thomas (then Dr.) Watson (1836-37), Dr. Walshe, and Dr. Cotton. (In Return 320 it is maintained that Dr. Cotton was of a different opinion 'a year before he died.')

"But Koch's recent discovery, together with the modern investigations into the infective properties of tubercle, open a new phase in the study of phthisis. The discovery, by Dr. Ransome, of bacilli in the condensed exhalations of patients in advanced phthisis, is also of considerable interest in connection with this discussion. The Collective Investigation Committee desire especially to draw the attention of practitioners throughout the country, who, they feel, have the best means of following closely the histories of their patients, to the practical problem which must be, to a great extent, solved by clinical observation, viz., whether phthisis is, or is not, communicable under certain condi-

tions from one person to another ; and, if so, what are the conditions under which this communication takes place.

“ It will be observed that the Committee, in asking for information as to the possible co-existence of family predisposition, are careful to distinguish between direct inheritance from parents or grandparents, and the collateral occurrence of the disease amongst brothers and sisters ; for it is often within the limits of possibility that, in the latter case, the disease may have been communicated to more than one member of a family from a common source.”

It having been brought to the notice of the Committee that the circular containing the questions cited had been overlooked by many members of the Association, it was again circulated with the issue of the Journal of March 3rd.

In response to these two issues the Committee have received 1078 returns.

A great number of these returns (673) were simple negatives, containing only the word “ No,” meaning that no case had been observed by the member making the return, but without any expression of opinion. These returns, admitting of no further analysis, have been set aside.

The remaining answers contained observations of some kind, and these have been classified as follows :—

Class 1.	Affirmative observers	.	.	.	261
„ 2.	Doubtful	„	.	.	39
„ 3.	Negative	„	.	.	105

The returns in Class I. containing the affirmative observations have been divided into the following ‘ groups ’ :—

- A. Returns of cases observed between *husband and wife only*, amounting to 158.
- B. Returns of cases observed between *members of the same family*—between parents and children, brothers and sisters, &c., as well as between husband and wife, amounting to 81.
- C. Returns containing cases observed between persons *unrelated*, as well as between members of the same family, amounting to 13.
- D. Returns containing cases observed between persons *unrelated only*, amounting to 8.

The Committee wished to publish and place before the Association the whole of these returns in full, but it was found that to do so would have required much more space than was at their disposal. They have, therefore, been obliged to use their discretion in condensing some of the returns and in omitting others. The returns that have been omitted in Class II. and III. must not be considered as of less importance than those that have been published; they have been omitted simply for want of space and because they resembled, more or less, those representative ones which have been selected for publication. Many of the returns in Class I. have been printed exactly as they reached the hands of the Committee, and it is hoped that the process of condensation which it has been found necessary to apply to many others will not be found to have led to the omission of any important fact.

Of thirty-nine returns from observers who were "doubtful," ten have been selected as fairly representative of that class, and published in the Appendix; of 105 returns from observers who appeared to answer in the negative, eighteen representative ones have been selected for publication. It is possible that some of the returns which have been classed as negative (*e.g.* Return 311) might more fitly have been included amongst the "doubtful," but we have felt bound to be guided by the strictly verbal form of the answers, and not to venture on any unauthorised interpretation of them.

The Committee desire to express their regret that it should have been necessary to omit any of these returns; they had all been prepared for publication, and it was only at the last moment found that they would require more space than it was possible to assign them. All the returns have, however, been preserved, and will be accessible to members of the Association.

It has not appeared advisable to the Committee to apply to these returns any strictly statistical method of analysis: such for example as the one so ably developed in Dr. Longstaff's communication.

It has seemed to them that a precise numerical method can only be satisfactorily applied to returns which have been obtained with the clear understanding that they are to be submitted to such a process of analysis; or to an investigation the plan and



details of which have been carefully devised with a view to this method of treatment.

A glance at the returns we have received will, we think, show that they are not adapted to such a process of estimation. Although many of the returns are remarkably precise and definite, there are others in which the observers have necessarily had to trust to their memories, and we meet with such statements as these—"I have seen more than one instance" (24); "I have seen several such cases" (47); "Certainly three or four cases" (62); "I can call to memory at least five or six cases" (72); "I have observed many cases" (105); "Suspected it in two cases" (117).

It will, however, be the duty of the Committee to propose to the Association a plan of further continued and contemporary investigation, to which a strictly statistical method may be fully applicable.

Out of the 1078 members of the Association who have returned answers to the questions issued on this subject at least 261 believe they have seen cases of phthisis which have originated in communication from one person to another; about 39 more have seen cases which have made them *doubtful* whether phthisis may not be so communicated; while 105 have offered facts and arguments which seem to them to negative such a view. One hundred and fifty-eight of the affirmative returns refer *exclusively* to cases observed between husband and wife. Communication between husband and wife is mentioned, together with other cases, in 34 of the remaining returns. So that 192 observers report cases of supposed communication of phthisis occurring between husband and wife.

Of the cases in which it is distinctly specified which partner conveyed it to the other, in 119 it is stated that the disease was transmitted *from* husband *to* wife; in 69 *from* wife *to* husband. It is distinctly stated in 130 of these cases that there existed *no family predisposition* to phthisis in the partner to whom it was supposed to be conveyed.

In 27 the family history has not been stated, in 7 it seems incomplete, and in 9 there may have been some predisposition.

In 32 returns reference is made to cases of supposed communication occurring between brothers or sisters, or between sisters



and brothers, and in 27 of these it is stated that there was *no family predisposition* to the disease, in 3 there was predisposition, and of the remainder there is no information.

In 18 returns cases are recorded occurring between parents and children, and in all but one there was *no family predisposition* discoverable. Cases are also recorded between step-parents and step-children, sisters-in-law and brothers-in-law, brothers and half-brothers, aunts and nieces, uncles and nieces and cousins, where there has been *no family predisposition*. Reference will subsequently be made to those remarkable records of instances where all or nearly all the members of a family have been carried off in rapid succession by phthisis.

Very few returns have been made of cases of supposed communication occurring amongst persons entirely unrelated to one another.

In 20 only of the returns received are such instances mentioned. As these have an important bearing on the question under consideration it may be as well to analyse them here in detail.

In Return 241, it is a servant who had devotedly nursed her master and mistress with phthisis: she died soon afterwards; "in her case there may have been some predisposition." In 242, it is a young man who "slept with" a phthisical friend, *no family predisposition*. In 243, it is a case between two friends. In 244, it is an apprentice who "slept with" his phthisical master; *no predisposition*. In 245, it is a young man who was "constantly" with the girl he was engaged to until she died of phthisis; *no predisposition*. In 246, it is a young girl who "was attending to" a case of phthisis in a male subject; *no phthisical history in the family*. In 247, "patient and nurse." In 248, it is a young maid-servant who nursed and "slept in the same bed" with her mistress, "who was phthisical," till her death. In 249, it is a young man who appeared to have contracted phthisis by "close contact with a fellow-workman who had phthisis;" *no predisposition*. In 250, "patient and nurse;" *no predisposition*. In 251, it is a young "healthy servant girl" who "slept with" a phthisical fellow-servant: *no predisposition*. In 252, it is a young lady who "slept in the same room" with her phthisical governess, and "never left her till her death;" *no family predisposition*. In 253, it is a female servant, whose duty it was "to empty and

clean the spittoons" of two phthisical patients twice a day; *no predisposition*. In 254, it is a young girl, "maid and companion" to a phthisical lady, whom she was "in the habit of sleeping with;" *no predisposition*. In 255, we have the somewhat striking circumstance of three young girls from three adjoining villages, apprentices to a phthisical dressmaker, "who took it in turn to remain in the house and sleep with her." In less than two years from her death they all died of phthisis, "although in the family history of each no trace of phthisis existed." In 256, it is "a perfectly healthy child," nursed night and day by a phthisical nurse; *no predisposition*. In 257, we have the patients in one particular ward of an asylum, the keeper of which was phthisical. In 258, it is a young woman who nursed a case of advanced phthisis; *no predisposition*. In 259, sailors in the same ship. In 260, it is "a young female who slept with a phthisical bed-fellow."

It will be observed that in all these cases there existed the same close intimacy as is found amongst members of the same family.

We have examined the returns to discover if we could obtain any precise information as to the period of time which elapsed between the assumed exposure to infection and the manifestation of the disease in the person assumed to be infected. In 92 returns we find no perfectly definite information on this point; in 48 we find that the disease became manifest *before the death* of the person or persons who were supposed to be the source of the infection; in 41 "*immediately*" or "*soon after*;" in 35 "*a few months after*;" in 26 "*a few weeks*;" in 24 the persons assumed to have been infected *pre-deceased* those who were assumed to be the source of the infection; in 20 the death of the person to whom the disease was supposed to be communicated occurred *within six to twelve months* of that of the person supposed to have conveyed the disease; in 11 *within twelve to eighteen months*; in 9 *within two years*; in 3 *within three to four years*; in 1 it is said "*a year or two afterwards*;" in 1 "*survived two years*;" and in 4 it is stated that the patient "*died after child-birth*."

Considering that these returns are, to a great extent, retrospective, greater accuracy than this could not have been expected.

In respect to the *course* followed in the cases supposed to arise

from communication, we find some kind of definite statement on this head is made with reference to 105 of the cases. Fifty-four of these are described either as "acute phthisis" or "galloping consumption," or "extremely rapid," or "rapid," or "very acute," or "quickly fatal," or as a "very short illness," or running its course "in a few weeks," or "in six or seven weeks," or "in a few months."

18	{ are described as running their course } within . . . . . }	5 to 12 months.
16	„ „ „	12 to 18 „
12	„ „ „	2 to 3 years.
3	„ „ „	3 to 7 „
and 2	„ as chronic phthisis.	

The very rapid course of so relatively great a number of the cases is noteworthy.

The occurrence of hæmorrhage is specially mentioned in thirty of the cases.

Bad ventilation is referred to in nineteen returns; and in two or three reference is made to the occupancy of "box beds" or "bed closets" in certain parts of Scotland (Returns 32, 166, 172).

Testimony as to *recovery*, after supposed communication, is borne in Returns 22, 55, 61, 75, 82, 103, 142, 164, 228, 233, and 246, and two of these cases (75 and 228) are in the persons of medical men, and reported by themselves.

The impression which some of these cases made upon the observers is worthy of note as bearing testimony alike to the rarity of the occurrence and the genuineness of the observation. In Return 22 we read of "One well-marked case in *forty years' extensive practice*." In 46 (and 82) "One case made a *strong impression* on my mind. It was *about eight years ago*." In 48, "One case forty or fifty years ago." In 60, "The case came under my notice *ten years ago*." In 183, "One case during thirty-six years of general practice;" and in others.

As remarkable records of a number of cases *occurring in succession in the same family*, without, in many cases, any inherited predisposition, the following returns are of special interest:—159. Three children; no case occurring after "the family of several children *left the house*." 165. Mother and three children.



169. Father and four children; "family history not known."  
173. Three children in succession. 177. Mother and two sons.  
178. Father, mother, and two daughters. 188. Two brothers and a sister. 193. A remarkable return, presenting many points of interest; notably the immunity of the father while the wife and nine children died of phthisis. 194. Mother, three sons, and a daughter; this and the Return 196, father, mother, son, and three daughters, are noteworthy from the manner in which the disease seemed to be introduced in both cases. 217. Six or seven brothers and sisters. 218. Three brothers and sisters, within sixteen weeks, &c. 280. Two sisters and a brother, within two months.

The following returns may also be noted as of much interest:—  
161, 162, 163, 167, 168, 172, 175, 176, 180, 187, 199, 204, 205, 228, 252, 253, 254, 255, 256, 257.

There is little for the Committee to note in the returns of Class II.; they fairly represent the condition of doubtfulness which must be present in many minds when considering this difficult problem. Return 262 well displays this state of hesitation. In 263 we have the evidence of a well-known provincial physician, after 50 years' experience, who bears witness to having seen many cases "which have appeared to have for the exciting cause communication with persons suffering from the same disease, but," he adds, "I do not remember any case so caused where there has been no hereditary disposition."

Return 265, as referring to a case occurring in a nurse in a hospital for consumption, is of interest.

A very pertinent reflection occurs in 268: "In a country practice it is almost impossible to decide between phthisis communicated and hereditary, as patients are generally nursed by members of the same family." Return 278 is again suggestive of the great difficulty of arriving at a final opinion on this subject.

The interesting record in Return 280 is well calculated to prompt the remark: "The history of the whole case is difficult to ascertain."

Returns 283 and 289 are valuable and suggestive contributions to this investigation.

Passing now to the negative returns of Class III., they present several points worthy of note.



In the first place we notice that while some of those observers express a *decidedly* negative opinion, others, as we have already remarked, might perhaps be more correctly classed with the "doubtful." For example, in 392, we have strongly negative testimony—"I never have observed a case of even probable transmission of phthisis from one person to another;" and in 302, "I have seen no such case;" and in 307, "I have never seen such a case." In 303 we have a more modified statement, "I am not aware that I have seen a case of phthisis directly communicated from one person to another without some hereditary predisposition to the disease, or family relationship between individuals concerned;" so also in 306, "have observed several instances of husband and wife suffering from phthisis, but have had *no direct proof* that the one received it from the other."

It would have been interesting if this observer had stated what he would in such a case consider to be "direct proof." Return 304 does not express any negative opinion, but it is of value as showing how by removing conditions which might favour communication, if communication be possible, the occurrence of phthisis in the army has been greatly diminished.

In 302 we see the influence of pathological doctrine or opinion, as this observer clearly regards phthisis as etiologically related to other "chest diseases."

305 is a good, simple, negative observation.

It is also deserving of remark that the cases cited in 309 bear little or no resemblance to the great majority of cases recorded in Class I., and would certainly not be claimed by these observers as cases of communication. The long intervals between these cases presenting a very great contrast to those recorded in that division.

On the other hand, Return 311 relates a family history which no doubt some observers would have returned amongst the affirmative class. In the thoughtful Return 315 it is suggested, "that what might have been called contagion has been a case where one cause has produced the same effect on many people in the same condition," and that they have "all been infected from one common cause;" thus while recognizing the operation of an "infective" cause, this observer doubts the possibility of this cause being conveyed from one person to another; a subject to which

we must presently return. He adds—"in the case of husband and wife I have never seen anything to arouse a suspicion that one has infected the other;" here again it would have been interesting to learn what this observer would consider sufficient "to arouse a suspicion."

316 is a representative of the simple negative observations of this class.

In Return 317 an observer of very considerable experience in connection with phthisis, observes, "cases of wives worn out by nursing consumptive husbands, and becoming themselves consumptive, I have many times noted, but in no case could I put the disease down to contagion *exclusively*." This seems to imply that "contagion" *may* have had *some* causative connection with these cases; and this is all that seems to be claimed by the majority of *affirmative* observers.

We have already called attention to case 320; it is important, as Dr. Cotton, to whom it refers, has been widely quoted as holding a strongly negative opinion. Return 329 presents a negative observation in perhaps as strong a form as is possible. The desire to accept any explanation of observed facts rather than that of "communicability" is exemplified in Return 339. Return 350 is remarkable as showing how the same kind of evidence will produce opposite conclusions in different minds, according to the "point of view" from which it is regarded. "Man, wife, daughter, and four sons living together. All four sons died in the last three years; father, mother, and sister apparently unaffected." The four sons dying in quick succession might have been cited by an *affirmative* observer as an evidence of communication, but the escape of the father, mother, and sister is here set forth as *negative* evidence. Finally, 393 is a good representative of a *wholly exceptional* experience. It is of much value as showing how widely *individual* may differ from *collective* observation.

In presenting to the Association the foregoing examination of the evidence furnished by the returns printed in the Appendix, the Committee have desired to draw attention to a few of the salient points which they present; but they especially desire to call the careful attention of all who are interested in this investigation to the Returns themselves, believing that they will well repay an

attentive study. While the Committee have considered it to be their duty to examine and classify, so far as was practicable, the evidence here collected, they do not think it incumbent upon them to pronounce a verdict. It is competent to all the members of the Association to act as jurors in this case; the evidence is before them, and they can form their own judgment. There are, however, a few considerations which present themselves to the minds of the Committee, and which they think should be weighed together with the evidence here set forth.

And, first, with regard to what is understood by "inherited disposition," or "family predisposition;" there would seem to be an idea in many minds that the fact of "inherited disposition" is incompatible with "communicability," but a very little reflection will show that this is not the case. It is obvious that the cause *communicated* may be the *exciting* cause, and the condition inherited the *predisposing* cause; so that predisposition and communicability instead of being antagonistic may be co-operative.

Attention is called to this in Return 193, one of the most remarkable of the series. "It does not follow," this observer remarks, "that because a parent develops phthisis his offspring cannot bear witness to the communicability of phthisis. If the affected parent is alive after the birth of the child there is possibility of communication."

It is also well to remember in this connection that we are not acquainted with any purely constitutional inherited disease which attacks and destroys several members of a family in rapid succession, as is recorded in several of these returns. The argument from analogy, whatever may be its value, is here certainly on the side of communicability. The fact also appears to have been lost sight of by those who have allowed their minds to dwell too exclusively on "family predisposition" as opposed to "communication," that the more communicable a common disease like phthisis might be, the more certainly would it be found frequently occurring amongst our ancestors. No one thinks of arguing that whooping cough is an "inherited" and not a "communicable" disease, because it is constantly found occurring amongst our parents and relatives. We make these observations simply to show that the question of "communicability" is independent of "inherited predisposition," and that the two things are in no way antagonistic.



Then again with regard to the influence of catarrhal attacks and irritating atmospheric conditions, such as the presence of dust and mechanically irritating substances, &c., in the air, which are regarded as causes of phthisis; this belief also is not inconsistent with communication. This is insisted upon in the thoughtful and suggestive Return No. 18. "My opinion is this. Phthisis is contagious when a mucous membrane is wounded, unhealthy, or even ill-nourished, but it is not easily communicated to a healthy subject in whom none of these conditions are found."

It should also be borne in mind that over-crowding and defective ventilation, which seem to be generally admitted to be predisposing causes of phthisis, would undoubtedly favour communication through the agency of an infective medium; the presence of such a medium under such circumstances, is suggested even by those who oppose the view of communicability. (Return 315.)

The *predisposing* causes of phthisis may, therefore, be various and multiform; but the *immediate exciting* cause may be specific definite, and uniform. This is what has been asserted recently to be the outcome of repeated pathological experiment and observations—observations, it is maintained, which have been submitted to the most conclusive and repeated experimental tests. It is here that pathological experiment crosses the line of clinical research, and its influence on the investigations with which we are concerned cannot be overlooked.

One fact these returns seem to establish beyond any question, and that is, that if phthisis is a communicable disease it is so only under circumstances and conditions of extremely close personal intimacy, such as persons sharing the same bed or the same room, or shut up together in numbers in close, ill-ventilated apartments.

The Committee, in conclusion, venture to express a hope that this first step in collective investigation into one of the most important questions connected with the etiology of phthisis will be of considerable value in leading to more open-mindedness in the discussion of disputed questions of experience and observation.

It seems perfectly clear, from a consideration of the evidence here collected, that many observers of undoubted capacity and



trustworthiness have observed cases which it has never fallen to the lot of many others to encounter.

It is, perhaps, equally certain that many observers of equal ability and carefulness have been led to draw different inferences from the observation of the same facts, and these differences of opinion may probably in many instances be traced to the existence in their minds of pre-accepted pathological doctrines.

Nothing could show more conclusively the value of *collective* investigation as opposed to *individual* impressions.

*The Sub-Committee is indebted to Dr. BURNEY YEO for the preparation of this Report.*

(Signed) DYCE DUCKWORTH, M.D.  
FREDERICK TAYLOR, M.D.  
W. J. TYSON, M.D.  
J. BURNEY YEO, M.D.  
F. A. MAHOMED, M.B., *Secretary*  
*to the Committee.*

NOTE.—The Committee have received several valuable suggestions, including one from Dr. Duncan Mackenzie of Glossop, with reference to the continuance of this investigation. The Committee propose, at an early date, to issue a Card, to be circulated amongst members of the Association, for the observation and record of contemporary cases; this card will embrace the whole subject of the Etiology of Phthisis.

# APPENDIX OF RETURNS ON THE COMMUNICABILITY OF PHTHISIS.

## CLASS I.

### AFFIRMATIVE OBSERVERS.

#### GROUP A.

*Returns of cases observed between husband and wife only.*

#### Section I.\*

NAME AND ADDRESS OF OBSERVER.	NO. OF RETURN (for identification only).
Weber, Hermann, M.D., F.R.C.P., 10, Grosvenor-street, W.	1
Whittle, Edward A., L.R.C.P., Duffield, Derby	2
Davies, H. Ll., M.B., C.M., Holywell, N. Wales	3
Althaus, Julius, M.D., M.R.C.P., 36, Bryanston-street, W.	4
Hudson, Robert S., M.D., Redruth, Cornwall	5
Few, W., M.R.C.S., Ramsey, Huntingdon	6
Balding, D.B., F.R.C.S., and Areher, H. R., M.D., } Royston	7
Murrell, William, M.D., 38, Weymouth-street, W.	8
Sutherland, Arthur, M.B., C.M., Invergordon, N.B.	9
Barry, D. P., M.D., Surg.-Major, Bury St. Edmunds	10
Hayes, Hawkesley Roche, L.R.C.P., Basingstoke	11
Hollis, W. A., M.D., Brighton	12
Munby, B. H., M.D., Aberdeen	13
Ellison, James, M.D., Windsor	14
May, G. Parker, M.D., Maldon	15
Kerswill, Bedford, M.R.C.P.E., St. Germans, Cornwall	16
Maevie, S., M.B., C.M., Chirnside, N.B.	17
Goodechild, J. A., L.R.C.P., Bordighera	18
Martin, James, F.R.C.S., Woodview, Portlaw	19
Walker, Alex., M.D., Putney	20
Oliver, J. F., M.D., Putney	21
Watkins, Robert Webb, F.R.C.S., Toweester	22
Quinlaw, F. J. B., M.D., Fitzwilliam-street, Dublin	23
Meredith, John, M.D., Wellington	24
Leachman, A. W., M.D., Fairley, Petersfield	25
Parette, James, L.R.C.P., Bristol	26
Suteliffe, John, M.R.C.S., Stalybridge	27
Flower, Fredk. J., M.R.C.S., Warminster	28
Welply, J. J., M.D., Bandon, co. Cork	29
Mathers, Adam A. C., M.D., Coleraine, co. Derry	30
Hunt, De Vere, L.R.C.S., Bolton	31
Wilson, R. M., M.D., Old Deer, Mintlaw, N.B.	32

\* This "group" has been divided into two "sections," the returns in the second section appearing not quite so definite as those in the first.

NAME AND ADDRESS OF OBSERVER.	No. OF RETURN (for identification only).
Mossop, Isaac, L.R.C.P., Bradford . . . . .	33
Evans, Charles Jewel, M.R.C.S., Northampton . . . . .	34
Biddle, Cornelius, L.R.C.P., Merthyr Tydvil . . . . .	35
Bain, W., L.R.C.P., Heaton Chapel, Stockpool . . . . .	36
Vawdrey, T. G., L.R.C.P., Handsworth, Birmingham . . . . .	37
Underhill, T. Edgar, M.B., F.R.C.S.E., Tipton Green . . . . .	38
Hodgkinson, Alex., M.B., C.M., Manchester . . . . .	39
Wade, A. Law, M.D., Somerset Co. Asylum, Wells . . . . .	40
Balfour, Thomas A. G., M.D., F.R.C.P.E., Edinburgh . . . . .	41
Moorhead, William R., M.D., Benburb, co. Tyrone . . . . .	42
Watson, George S., M.R.C.S., Tunbridge Wells . . . . .	43
Bean, Dex, L.F.P.S., Moseley, Manchester . . . . .	44
Sims, J. Marion, M.D., 12, Place Vendôme, Paris . . . . .	45
Barnes, Henry, M.D., Carlisle . . . . .	46
Bradley, Daniel, L.R.C.P., Dndley . . . . .	47
Cade, Thos. Chas., Spondon, Derby . . . . .	48
Baird, Alex., M.D., Perth . . . . .	49
Diekson, Walter, M.D., 32, Belvedere-road, Upper Norwood, S.E. . . . .	50
Duneanson, T. J. Kirk, M.D., 22, Drumsheugh Gardens, Edinburgh . . . . .	51
Somerville, Robert, M.D., Galashiels, N.B. . . . .	52
Skinner, Edward, L.R.C.P., Sheffield . . . . .	53
Smyth, Spenceer T., M.D., Honor Oak, S.E. . . . .	54
Martin, John W., M.D., Sheffield . . . . .	55
Pileher, W. J., F.R.C.S., Boston . . . . .	56
Timms, Godwin W., M.D., 9, Wimpole-street, W. . . . .	57
Campbell, Arthnr James, M.B., Mablethorpe . . . . .	58
Wallis, Frederie M., M.R.C.S., Barraek Hall, Bexhill . . . . .	59
Stear, Henry, M.R.C.S., Saffron Walden . . . . .	60
Palmer, Charles, M.R.C.S., Great Yarmouth . . . . .	61
Napper, A. Arthur, M.R.C.S., Cranleigh, Surrey . . . . .	62
Miller, J. D., M.B., 1 St. Ann's-road, Notting Hill, W. . . . .	63
St. George, George, L.K.Q.C.P.I., Lisburn, co. Antrim . . . . .	64
Barron, Alexander, M.B., University College, Liverpool . . . . .	65
Brandt, G. H., M.D., Oporto . . . . .	66
Newcombe, Charles F., M.D., East Twickenham . . . . .	67
Candler, John, M.R.C.S., Haleston . . . . .	68
Bodkin, William, M.D., Chelmsford . . . . .	69
Sellers, William, L.R.C.P., Sileby, Loughborough . . . . .	70
O'Connor, Bernard, M.D., M.R.C.P., 40, Brook-street, W. . . . .	71
Lyster, Charles George, L.K.Q.C.P.I., Kilkenny . . . . .	72
Greenwood, jnn., M., L.R.C.P., Dalston . . . . .	73
George, Henry, M.R.C.S., North Thoresby, Louth . . . . .	74
McDonagh, James A., F.R.C.S., Hampstead-road, N.W. . . . .	75
Wylie, William, M.D., Skipton . . . . .	76
Spry, H., Truro, Cornwall . . . . .	77
Goyder, David, M.D., Bradford . . . . .	78
Bernard, Walter, F.C.P.I., Londonderry . . . . .	79
Eyton-Jones, F., M.D., Wrexham . . . . .	80
Hollis, Alfred, M.D., Freshwater, I. of W. . . . .	81
Dawson, W. H., M.R.C.S., Great Malvern . . . . .	82
Napier, A. D. Leith, M.D., Abbeylands, Dunbar, N.B. . . . .	83
Crutchley, Henry, M.D., Alsager . . . . .	84
Abbott, C. E., L.K.Q.C.P.I., Braintree . . . . .	85
Lee, Francis B., F.R.C.P.E., Heekmondwike . . . . .	86
Holden, J. Sinclair, M.D., Sudbry . . . . .	87
Rawle, Francis, M.R.C.S., Titchfield . . . . .	88
Thomson, W. Sinclair, M.D., 40, Ladbroke Grove, W. . . . .	89
Williams, Miles M., L.R.C.P., Wheelton, Chorley . . . . .	90
Jamieson, James, L.R.C.P., F.R.C.S.E., 6, Buccleugh-place, Edinburgh . . . . .	91

NAME AND ADDRESS OF OBSERVER.	NO. OF RETURN (for identifica- tion only).
Fowler, Trevor, L.K.Q.C.P.I., Epping . . . . .	92
Bryan, John M., M.D., F.R.C.S., Northampton . . . . .	93
Smith, J. Blaikie, M.D., Aberdeen . . . . .	94
Allen, W. Hamilton, L.R.C.S.I., Bardney . . . . .	95
Prescott, A. Knight, M.R.C.S., Surg.-Major, Muttra, N.W.P. India . . . . .	96
Hinds, James, M.B., Halstead . . . . .	97
Moore, Henry Gage, M.D., Ipswich . . . . .	98
Oates, J. Harrison, M.R.C.S., Dewsbury . . . . .	99
Hunter, George, M.D., F.R.C.S., Llanthgow . . . . .	100
Prytherch, John, M.D., Liverpool . . . . .	101

1.—Since my communication to the Clinical Society (Trans. vol. vii.) I have observed two cases.

*Case 1.* In 1875. No relationship excepting that of wife to husband, the disease having been apparently communicated by the husband to the wife. Age at death 25, duration of disease about 10 months, development after second child-birth. No family predisposition.

*Case 2.* January, 1878. No blood relationship excepting that of wife to husband, the former having been taken ill soon after the husband's death. Age at death 26, rapid consumption commencing with hæmorrhage.

Two maternal aunts of this woman had died of phthisis.

NOTE.—Although the number of cases of apparent communication is small, my conviction of the communicability has been strengthened since the communication to the Clinical Society.

2.—One case in which phthisis was communicated by a woman to her husband.

In the autumn of 1879 I was called to attend a young married woman aged 21, who was suffering from tubercular phthisis, with a strong family history of this disease. She died in about three months. About six weeks after her death I was called to attend the husband, who, on examination, exhibited unmistakable signs of tubercular phthisis. He was attended by his club doctor, and died in about four months and a half of acute phthisis. I saw him before his death.

In this case the man was strong and healthy, and there was no history of phthisical disease in the family. He was a labourer, and against my advice slept in the same bedroom with his wife during her illness.

3.—In 1872 T. S. F., bank accountant, died of phthisis in the month of April. His wife, who had been his sole attendant and nurse during his illness, but in whom there was no hereditary predisposition to the disease, died of phthisis in the month of November following.

4.—One case in July, 1871.

A German gentleman, æt. 40, had advanced disease of the left lung, which he had apparently inherited from his mother. He had married a perfectly healthy woman, of good stock, three months before I saw him. The wife died of consumption the following February (1872), and her husband survived her two months.

There was no blood-relationship between the two.

5.—One case, survivor at present under my care. Husband died on 9th December, 1882, aged 31, and it would appear that he contracted the disease from his wife, who still lives, but in advanced phthisis. Both her lungs have cavities, and her sputum contains broken-down lung tissue (yellow elastic) and swarms of the bacillus tuberculosus.

Husband's name, H. P.; occupation, clerk. Comes of a healthy family. His father, mother, and all his brothers and sisters, eleven in number, survive, save one who died suddenly in California. All are grown up, in good health. Many have



families, and their children are healthy. I know his parents and many of his brothers and sisters, and no finer, healthier, or better-formed family is in the neighbourhood.

Wife of a very consumptive family. Father, a sailor, was drowned; mother died at 59 of bronchitis; but all her brothers and sisters died of consumption, and she alone survives.

They were married ten years ago; four children have been born. The two elder survive, but are delicate; the two younger died at 13 months and 7 months of tubercular meningitis. Mother had an attack of pleurisy four years ago, was nursed by husband. She had an attack of hæmoptysis about three years ago; dates are forgotten. During this illness they slept in a warm, close room. He began to fail in the summer of 1880, and during November, 1880, he was operated on for fistula *in ano*. This never healed, but during the summer of 1881 he *spat blood*, and was sent back to Cornwall to his native air.

6.—One well-marked case about two years ago.

A discharged soldier, confined to his bed with phthisis for many weeks; the bedroom small, low, and miserable, about twelve feet by eight. Weather, cold. Wife strong and healthy; her parents also. She slept with her husband nearly up to his decease, became phthisical a few weeks afterwards, and died in a few months.

Not any of her relatives phthisical, and the woman herself a remarkably fine, strong, healthy person previously.

7.—One case. T. W., a farmer of 30, first came under observation June 17 1879. On examination, apices of both lungs found diseased. Got gradually worse, and died of phthisis March 22, 1881. Wife of above, 28, of healthy family, and mother of two healthy children, was quite well when husband first came under observation. On April 13, 1880, first applied for advice, found seriously ill, and died of phthisis July 5, 1880, eight months before husband.

I have not been able to make out any phthisis in the family of the wife.

8.—Case observed at Royal Hospital for Diseases of the Chest in 1879.

Lizzie E., aged 26.

Family history:—Father died of fever, no cough, no chest disease. Mother living, asthmatic for twenty years, but nothing else. Three sisters alive and well. No history of phthisis in the family. Was in good health at time of marriage.

Husband's family history:—Father died of bronchitis. Mother died of consumption. Two brothers suffering from advanced consumption.

Married in 1878. In July husband broke a blood-vessel, became consumptive, and died in February, 1879. He expectorated a great deal, and wife nursed him and slept with him the whole time. She had no cough at the time of his death, but shortly afterwards she was seized with cold shivers, which lasted three days. A cough then appeared, with night-sweating and loss of flesh. Nine months later she was under treatment for consumption, the physical signs indicating breaking down at the apices of both lungs. The case was regarded as an example of phthisis of contagious origin. The wife suffered no privation after her husband's death, and was not exposed to cold.

9.—One case in 1875.

A tailor had a phthisical wife, in whose family several cases of phthisis occurred. In her case the disease ran a course of three or four years. Meantime her husband, who was not robust, but in whose family history phthisis had not, as far as I am aware, showed before, contracted tubercular disease of the lung, and died before her by more than a year. Their rooms were very close and warm.

10.—For many years I have considered that there is, under circumstances of close contact and cohabitation, some degree of contagiousness or infectibility of phthisis. The following cases came under my observation when I was in private practice at Twickenham, and made a great impression upon me.

Case 1. Mr. N., residing in Twickenham, but in a first-class business in Piccadilly, had a wife in chronic phthisis. He was a delicate man, aged about 30, and soon became phthisical himself. He overworked himself, and the disease ran a rapid course, terminating suddenly by rupture of a large pulmonary vessel. Mischief went on more slowly in the wife, aged about 30, and she died within twelve months of her husband.

*Case 2.* Mr. M., a builder's foreman, ago about 33, had a consumptive wife, ill for about three years. He was a remarkably fine man, and had never previously had any ill-health. Towards the close of his wife's sickness he became delicate suddenly. Very soon after her death all the symptoms of phthisis set in, and he proved an acute case, for he died shortly.

*Case 3.* Mrs. C., always a delicate young woman, developed phthisis, and was most sedulously tended and cared for by her husband throughout her illness of about eighteen months' duration, when she died. Mr. C., about 30, her husband, had always been a fine healthy man, very active in business, until near the close of his wife's lifetime, when he began to fail. A few months afterwards phthisis became manifest in him, and he died within a year of his wife.

No hereditary taint or other predisposing cause could be shown in his case. Nothing beyond the constant communication with the wife.

11.—One well-marked case in 1869.

A woman, about 26, in good health, family history on both sides good, nursed her husband, who was suffering from pulmonary consumption. Six months after his death symptoms of pulmonary consumption developed themselves, and she died of pulmonary phthisis within twelve months after the death of her husband.

12.—One case. Whilst casualty physician at St. Bartholomew's Hospital I saw and prescribed for a man, who, at the time, about ten years ago, was suffering and subsequently died from subacute tuberculosis of lungs. His wife, who had been previously a healthy woman, with no family history of lung disease that I could obtain, attended shortly after the husband's death, with acute pulmonary phthisis, from which she soon afterwards died. She always affirmed that she had "taken her husband's breath" while she was nursing him.

13.—One case in 1878, that of a lady whose husband died of phthisis. She was his nurse, and for several weeks hardly left his room, constantly attending to him night and day. She had galloping consumption, and died in little more than a month after her husband. Neither grandparents, parents, sisters, brothers, aunts, nor uncles had phthisis, but some of her cousins had.

14.—One case in 1879, which suggested to me the possibility that phthisis was communicated by a husband to his wife.

Two brothers of a consumptive family died of phthisis. The one who died last was married. His wife, who survived him two years, and who was not, as far as appeared, of a consumptive family, died also of phthisis.

15.—One case especially in which I believe the disease was contracted by the wife from the husband.

Husband, chronic phthisis, about four years' duration, died in December, 1856. Assiduously nursed by wife of a robust constitution, who slept with him until within a short period of his death. Symptoms began to develop in her the June following. She died November, 1857, æt. 46.

Wife's parents lived to 78 and 76 respectively. She was one of fifteen children, eleven of whom lived beyond 60. Most of them reached advanced age. One lady now living, aged 92, in good health. Of the four who died under 60 the earliest death occurred at 37. None but the subject of the present notes died of pulmonary consumption.

16.—S. S., private Royal Marine Light Infantry, was invalided for phthisis, and returned to his home, his wife then being in good health, and having no history of hereditary phthisis. He gradually became worse, and about twelve months after his return his wife contracted phthisis, evidently from him, which ran a rapid course, ending in her death, October 9, 1881, three months before her husband.

17.—One case in 1873, at Shotts, Lanarkshire.

The husband died after a lengthened illness of phthisis, leaving his wife pregnant. Just a few months before his death the wife manifested pectoral symptoms of a suspicious kind. During the pregnancy these made no advance, but afterwards



developed rapidly, and ended in death from tuberculosis, about four months after the birth.

The husband was a coal miner; there was no other predisposition.

**18.—Case 1 (1878–9).** Communicated from wife to husband. I was informed by various members of the family that no relative, or even connection, had ever suffered from phthisis, while the patient was in perfect health at the time of his wife's death, occupied the same chamber till the day before, and showed symptoms about eight weeks later.

I have seen several other cases in which I had no doubt that contagion had occurred, but none so marked or unimpeachable as the one mentioned above.

**Case 2.** A lady, aged 34, of consumptive parents, first became phthisical six or seven years ago, and came to the Riviera, where she has since resided during the winter. During the first two years she was nursing a sister, who died of the same complaint, and she made little or no gain herself. Since her sister's death she has been fairly active and well, and a very large cavity in the left lung has in great measure cicatrized. She has always had slight morning cough, and has once or twice suffered from hæmorrhage, though not to any great amount. Some four or five weeks back she undertook to nurse a bad case which had recently arrived. About three days later her cough increased to an alarming degree. She became very febrile, and the right lung, which had before been comparatively free from signs of disease, became greatly congested. Isolation from her patient, with a free use of disinfectant inhalations, subdued the symptoms, which had caused me the very gravest apprehension, in an astonishingly short time, and she is now in her usual condition.

I could multiply similar cases indefinitely, but instances must be within the knowledge of most practitioners, where members of a phthisical family have been themselves attacked while attending upon sick relatives, and also cases where previously healthy persons, who have been debilitated by syphilis or other causes, have become phthisical when brought into contact with the contagious.

**NOTE.**—My opinion is this, "Phthisis is contagious when a mucous membrane is wounded, unhealthy, or even ill-nourished, but is not easily communicated to a healthy subject in whom none of these conditions are found." In the case which I have forwarded upon the form, the only circumstance which I could learn bearing upon my view, was that, at the time of his wife's death, my patient, a robust fellow of 27, was suffering from slight sore throat. This throat continued to grow more irritable for some weeks afterwards, and about eight weeks later severe hæmorrhage from the throat came on, causing great debility. After this, marked phthisical symptoms set in with great rapidity, and he was sent abroad. Here, in spite of one or two slight attacks of pulmonary hæmorrhage early in the season, he did well under antiseptic treatment, and recovered much of his former health. About a year and a half later he had a severe attack of hæmorrhage in England, and sank rapidly.

I relate this case first as it stands alone in my experience, but I have witnessed many cases of what I believe to be second contagion. Numerous cases in which recovery, partial or apparently complete, has taken place have relapsed under my observation, when brought into contact with other cases of phthisis. I have noticed that this has been most frequently the case where the general health has remained feeble. As an illustration I have given one case which has been under my own care during the last few weeks.

**19.—**A clergyman, married at 27, had tubercle at the upper part of the right lung, and hæmoptysis. His wife was a healthy young woman, in whose family phthisis was unknown. She contracted tubercular disease of a very acute form, and died in seven weeks after first complaining, eighteen months after marriage. He lived seven years afterwards, and again contracted matrimony, but having left my district I know nothing of the result.

**20.—**A. S., a cousin of mine, aged 30, married a young lady who was suffering from consumption. She died six months after marriage, and in six weeks from the death he contracted the disease and died.

There was no consumption in A. S.'s family. The wife's family were decidedly tuberculous.

21.—A. B., a cook, widow, aged 37, one child of 5 years old. Married her husband when he was said to be dying of consumption. He lived a few months. She became phthisical.

I knew the family well. There was no tubercular taint on either side. She became an in-patient of Durham County Hospital, where she died.

22.—One well marked case in forty years' extensive practice, twenty-five to thirty years ago. The husband of a lady who died of phthisis. Neither his father, mother, nor only brother, had phthisis. Neither of the sons, nor daughters (I have lost sight of two daughters) have suffered from phthisis.

The subject was a robust farmer in a very healthy village, about 40 years old. He was attended by the late Dr. Kerr of Northampton and myself. He had no history of pneumonia or bronchitis. He became fairly convalescent but with some dulness remaining in both lungs, and resumed work in less than twelve months. He married a second wife, and begat sons and daughters. He died after a few years of renal disease and chronic cystitis.

23.—I came upon an undoubted case of communicability of phthisis pulmonalis from husband to wife this morning at St. Vincent's Hospital.

This woman, Bridget O'Loughlan, lives at 1, Maekey's Terrace. Mother and father in perfect health. There is no phthisical history in her family.

Her husband, a pensioner discharged from the army five years ago for pulmonary deliacy, fell into acute phthisis in January, 1881, and died on April 9 following. She had been married three years and three months, and had two children; both are dead, one of water on the brain, and the other of convulsions, possibly due to tubercular meningitis.

Till her husband's last illness she never had a day's ill-health. Towards the end of it she sickened, and immediately after his death had hæmoptysis, night sweats, &c. She is now in advanced consumption, dulness under the clavicle, moist crepitus, hectic emaciation, &c.

I have observed five other cases of undoubted communication of phthisis pulmonalis from one individual to another—husband and wife.

I could not in any of these five cases trace any family predisposition in the party apparently affected. I may add that I have seen many cases of consumption occurring in both husband and wife which did not appear to bear on the question, as there was a phthisical history in both sufferers.

NOTE by Dr. Mahomed.—I have seen this case and could trace no family history. It is certainly phthisis.

24.—I have seen more than one instance but I can only specify one now, as I have no notes of the others.

Case of wife and husband. Husband clerk and traveller for a firm. Lived in a damp thatched house. Wife showed signs of phthisis in 1875. They removed in about a year to a drier and better house, but phthisis persisted, and she died in January, 1877. Husband had now begun to show undoubted symptoms of phthisis, became worse, and died of it in March, 1878. He persisted in occupying the same room, and nearly always the same bed as his wife. His illness did not begin until long after his wife's, and after they had left the damp house.

There was no family predisposition on either side, and no brothers or sisters have suffered from phthisis pulmonalis, the youngest child of the deceased persons, born, it is suspected, when the mother was beginning her illness, has been delicate (serofulous sores on neck), but she is growing and likely to do well. The other (eldest), born when parents were well and strong, has always been healthy and hearty.

25.—W. B. C., a painter, died May 10, 1882, aged 41, of tubercular phthisis.

His wife, aged 38, was a stout healthy-looking person, with no hereditary predisposition to tubercular disease on her father's or mother's side. Her mother is alive and well, aged 72. Her father died at 50, but not, they say, of consumption. Her mother says she never had a day's illness till her last and fatal one. She died on October 7, 1882, of phthisis, having failed about four months before and kept her bed five



weeks. I may mention that *she* was not under my care, but under that of the Poor Law Medical Officer, from whom I got the information as to her disease.

**26.**—The following case did not come under my personal notice, but, being so conclusive, and I one of the relations, I must record it.

F. B., surgeon, married a phthisical wife, who, having given birth to two children, died of phthisis in childbed with the third in January, 1847. F. B. died of phthisis in October, 1848, aged 34.

F. B. was apparently in good health up to the date of his wife's death. The case appeared to commence with hæmoptysis, and ran a rapid course in three months.

The two children are now living, one is 41, the other 37 years old.

F. B.'s father died at 85, mother died at 84; three sisters are now living, aged 64, 62, and 60; one brother, a surgeon, was killed at 45.

No other relation ever died of or had any symptoms of phthisis. A post-mortem was made by the only brother, a surgeon.

**27.**—One case, February, 1882.

A girl, in good health, with a family history showing no trace of phthisis, married two or three years ago a man who afterwards suffered, and is at present suffering, from chronic phthisis. In January, 1882, she exhibited signs of incipient phthisis, which rapidly developed after she aborted, about February 5. She died on April 17, 1882.

**28.**—I have seen two distinct cases communicated from husband to wife.

In one there was no family predisposition. The wife was a strong healthy woman at the death of her husband, but within a few weeks she had symptoms of pulmonary phthisis, and died within three months of her husband.

In the other case there was a strong hereditary predisposition.

**29.**—Two cases.

*Case 1.* A very healthy young man, whose relatives had no predisposition to the disease, showed signs of pulmonary phthisis a few months after his marriage to a woman who was suffering from the disease. Her case was progressing slowly, his rapidly. Both died on same day.

*Case 2.* A widower married a woman who showed signs of phthisis shortly after their marriage. In about eighteen months she died. A little before her death he got ill, and is now under my care suffering from same disease.

None of his relatives, or of his children by first marriage, showed the least predisposition to the disease, and he was strong and healthy when married for the second time.

**30.**—Case of husband apparently infecting wife.

The husband presented marked physical signs of phthisis, of which he subsequently died. His relations were markedly phthisical: so are his children. His wife was very healthy, with no trace of phthisis in her family history. Six or seven years after marriage the wife began to complain of chest, and soon presented well-marked signs of pulmonary phthisis. Destruction of lung tissue was very rapid, leading quickly to a fatal result. She died shortly after her husband.

**31.**—One case.

W. S. L. was married in October, 1869. His first wife died in April, 1878, of phthisis pulmonalis. Soon after her death he got ill with the primary symptoms of phthisis. He improved in health, and then married a second time a healthy woman, who died in 1880 of phthisis. He is still living, or rather dying, in the last stage of phthisis. Each wife left a child, and they also have all the constitutional symptoms of this disease.

**32.**—I have a case which appeared to my mind pretty conclusive.

T. M., farmer, in fairly good circumstances, died of tubercular phthisis as certified by my predecessor, Dr. J. C., who attended him during a long illness. The death took place August 2, 1872. I commenced practice here in 1875, when Dr. C., amongst other patients, handed over to my care Mrs. M., who, he remarked at

the time, had been infected by her husband, as she slept with him during the whole course of his illness in a small bed-closet. Mrs. M. died of tuberculosis on December 5, 1876.

Her family history was good. Her mother survived her four years, and was 82 at her death. Her father was an old man, and her two sisters and brother live in sound health in this parish, being engaged in agricultural pursuits.

J. M. was 38, Mrs. M. 35.

**33.—Case 1.** Husband to wife. James S., 43, admitted to Edinburgh Infirmary, December 18, 1868, suffering from phthisis pulmonalis, and died there.

He married in 1866 S. S., aged 21, a very healthy girl, whose parents, brothers, and sisters are living and well. No trace of consumption in her grandparents or their offspring. Admitted to Edinburgh Infirmary with physical signs of phthisis January 17, 1869, having been for two months attended at her home by me as dispensary patient. Discharged somewhat improved after a month. Again became dispensary patient. Died in early part of August, 1869.

**NOTE.**—The man was nursed by his wife, and she slept with him up to the time of his admission. The room in which they lived was *badly ventilated*, being the middle flat or storey of a house in a close or court off the High Street in Edinburgh. The stench in the place was at times very bad. She had not had sufficient nourishment for some little time previous to and after her husband's admission to the infirmary. Her mother stated that up to 2 years of age she was a delicate child, and had whooping cough, but up to the time of her marriage was in perfect robust health. Her husband was absent from her for fifteen months, when two months married; and when he returned, consumption was established, she being at that time in good health.

**34.—February, 1883.** Husband and wife. Husband died at end of 1882, after suffering from phthisis for about three years. Both parties always occupied the same bed. Wife now the subject of advanced phthisis in one lung.

No family predisposition on her side. Both parents now living and in good health. No history of phthisis in any members of her family.

**35.—One well-marked case.**

Wife died of phthisis pulmonalis on May 11, 1881. Husband soon after began to complain of cough, &c., and he died on Jan. 8, 1882, of phthisis pulmonalis.

Cannot say as to brothers and sisters, but both parents on husband's side alive, and each about 70 years of age.

**36.—**About four years ago G. C. died of phthisis. His wife, previous to his illness, always enjoyed the best of health. Her family history was excellent. No member of the family suffered from consumption. She died about two years after her husband from phthisis.

**37.—**Case observed in 1875. The patient lived at Deptford. The disease was communicated to the patient, a man between 20 and 30 years of age, by his wife, who was at the time in an advanced stage of pulmonary phthisis.

There was no family predisposition whatever to account for its development. None of the patient's relatives, as far as could be traced, had died of any disorder even resembling pulmonary phthisis. The patient himself, a very intelligent man, attributed his complaint to contagion conveyed by means of his wife's breath.

**38.—**Mr. J. H., aged 41, married, died of pulmonary phthisis in Dec., 1881. His mother and other relatives had died of phthisis. His wife was a healthy woman, with no hereditary tendency to phthisis. She nursed him most carefully for four months before death, always sleeping in the same room. She died of pulmonary phthisis in June, 1882, exactly six months after her husband.

Some years ago, when travelling through country districts in Spain, I found a common notion prevailing among the lower orders that phthisis was contagious. They never used a room where a person had died of phthisis, without first thoroughly disinfecting it.

39.—*Case 1.*—Eleanor A. applied at Hospital for Consumption and Diseases of the Throat, Manchester, suffering from pulmonary phthisis. Age 34. Husband just died of pulmonary phthisis, during which illness she attended him.

Family history. Father and mother quite healthy; has three brothers, also in good health. Knows of no relations having died of consumption.

*Case 2.*—June 2, 1877. Jane B. applied at Hospital for Consumption and Diseases of the Throat, complaining of cough and spitting of blood. Husband died of consumption of seven weeks' duration, during which illness she nursed and slept with him.

No family history of phthisis.

*Case 3.*—On Nov. 18, 1878, Ellen K., 28, applied at Hospital for Consumption and Diseases of Throat. Was found suffering from second stage of phthisis pulmonalis. Husband died of consumption four years ago; was nursed by E. K. for ten months, who slept with him. She commenced to be ill soon after the husband's death. Was never ill before since childhood.

Family history. Mother alive, 70; father died of obstruction of the bowels, aged 40. No relative on either side has died of consumption. Has four sisters, all living and very healthy.

40.—In 1872 I attended a man suffering from phthisis in Ryde, Isle of Wight. His wife appeared perfectly healthy. I know nothing now of his family history, and have lost my notes. I repeatedly warned the wife not to sleep with him, but she did so in spite of my warnings. In 1873 the man died. The wife then went to live with her own relatives, all of whom, as far as I could discover, were healthy. In a few months she developed symptoms of phthisis, and died of galloping consumption.

I heard of no case of phthisis in the wife's family.

41.—About 1856 a young man, in delicate health, married a young woman who had previously enjoyed good health. I rather think he was phthisical before marriage, but at any rate that disease was manifest shortly after that event. In six months after marriage he died of the disease at the age of 23. During this illness, except for about two weeks before his death, when he was in the Royal Infirmary, his wife was constantly in attendance, and slept with her husband without adopting any precaution against inhaling his breath. Phthisis began to show itself in her during this attendance, and on the day of his funeral she was for the first time attacked with hæmoptysis. The whole duration of her illness was eight months, and she died at 21.

There was no predisposition traceable on the side of either her father or her mother. Two brothers died, but neither of them had phthisis; and her sister, who is alive, is free from any indication of that disease.

NOTE.—It is only right to mention that the room in which the young couple lived was on the ground floor and damp; but, on the other hand, it was the house in which her father, and mother, and members of her family were living, who never suffered from phthisis.

42.—*Case 1.*—About five years ago. Wife to husband. Aged 28 and 30 years (two children). The wife fell into consumption, and died. Just before her death the husband was seized with a cough, and soon after exhibited the symptoms of phthisis, which carried him off two years later. There was no hereditary predisposition to the disease on his part. His mother died at an advanced age, and his father is still living, aged 80.

*Case 2.*—Three years ago. Husband to wife. The former inherited a predisposition to the disease; the latter, as far as could be ascertained, did not. The husband was suffering from phthisis for three years before his death. A few weeks after that event his wife was found, upon examination, to be suffering from the same malady. She succumbed to it eventually, after an illness of two years. The husband's age at the time of his death was about 30, and his wife's 38. (They had a family of four children, one of whom, a girl of 17, who slept with her mother, has lately fallen a victim to the same disease. I think it probable that the last case was also one resulting from infection; but as hereditary predisposition to the disease existed, its occurrence may have been simply a *post hoc*.)

43.—A young woman, æt. 24, with hereditary history of phthisis, died under my



care, in 1874, of chronic pulmonary phthisis. Her husband, a fine, healthy young labouring man, with no family predisposition to the disease, nursed his wife most carefully and assiduously during her illness. He began to show symptoms of pulmonary mischief shortly after her death, and died of phthisis in little more than a twelvemonth.

This is but one instance out of several in which I was much impressed with the apparent communicability of phthisis from person to person. They were all cases among cottagers where the rooms were small, the ventilation bad, the food scanty, and the soil clay, conditions conducive to the development of phthisis under any circumstances: but the primary cases all showed hereditary predisposition, the secondary all occurred in healthy attendants on the primary.

44.—About 21 years ago. I saw a case in which the wife of A. W., aged 33, died of phthisis, she being of a decidedly consumptive family. Her illness lasted about 18 months. Up to within 6 or 7 months of her death her husband had enjoyed good health. He then began to show symptoms of phthisis, and died in about 9 months after his wife. His family was well known in the district, and not a single member of it had died of the disease. Since that period I have seen many cases which have quite convinced me that the disease is frequently communicated.

45.—For nearly forty years I have believed in the communicability of consumption.

I resided in Montgomery, Alabama, from 1840 to 1853, was then a general practitioner, and had an extensive town and country practice. And during these thirteen years I can now recall but one case of confirmed consumption in my own practice. Indeed consumption was rarely met with at that time in that section of country.

My belief in its contagiousness is based on the following facts:—

Mr. A., 33 years old, living in Lowndes county, 25 miles south of Montgomery, died of consumption in 1842. His widow, aged 30, had a troublesome cough at the time of her husband's death, which gradually grew worse; and in 1844 she married Mr. B., a widower of 45, having a grown son residing with him within five miles of Montgomery. Soon after the marriage of Mrs. A. and Mr. B. her cough increased rapidly, within a year she had all the symptoms of confirmed consumption, and during the next year she died of it. Some time before her death Mr. B. showed signs of the disease, and in three years he and his son both died of it.

This was in a climate and a country where consumption was at that time almost unknown.

I did not know Mr. A.'s antecedents, but I knew Mr. B. very well, and was the physician of his brother's family. They had no family history of consumption. They enjoyed good health generally, but, in common with their neighbours, they suffered occasional attacks of malarial fevers in the autumn, and other climatic diseases.

46.—One case made a strong impression on my mind. It was about eight years ago. The husband had hereditary tendency to phthisis; was a labourer; lived in a one-roomed cottage on a couch; he was many months labouring under the disease. Before his death, his wife, who had no hereditary tendency, developed tubercle in the lungs, and quickly followed him to his grave.

47.—I have seen several such cases. They were all cases in which either husband or wife died of pulmonary phthisis, and then the survivor developed the disease.

The most recent instance was one in which the husband had died of phthisis, and the wife, who was my patient, had advanced phthisis, with cavities in the lungs, two years after his death. There was no relationship between them before marriage, and the wife's family appeared free from predisposition.

In my neighbourhood, where there is a good deal of phthisis, it is popularly believed to be contagious.

48.—One case, forty or fifty years ago, a man without any family tendency to phthisis, married into a family with decided predisposition to this disease. The wife died of the disease, and he soon followed. From his general health and family connection, I should have thought him the last man to die of phthisis.



49.—1872. Husband and wife; latter first affected. Wife's family riddled with consumption; husband's healthy, but number very limited. Wife's disease ran a chronic course; husband's quickly fatal. Husband a miner.

50.—Wife, a healthy young woman, and of a healthy family, died of phthisis thirteen months after her husband, who had died of rapidly progressive consumption. She was pregnant at the time of his death, and gave birth to a child which only lived a few hours. This is the only case in my knowledge, among many of wives sleeping with phthisical husbands in their last illness, of the wife appearing to catch the disease, and here it seemed to be through the fetus.

51.—One very marked case in which the husband died of tuberculosis, contracted from his wife, who lingered in the disease from eighteen months to two years. The husband died in less than a year after his wife's death. They were both young, and had a family of three children very quickly.

The wife's father, one brother, and two sisters, died before her of consumption. Other children of the family had died in infancy of tuberculous disease. Her mother was healthy, the husband's family had no ascertainable tendency to phthisis. After his wife's death, he, being then in broken health, took to drinking and soon died.

Another case. Husband contracted consumption from wife. Husband now at Davos Platz; wife at Mentone.

Strong tendency to phthisis in wife's family, both on father's and mother's side.

I consider his family strumous. His father and mother well and apparently healthy. Taint of struma in his brothers and sisters.

52.—A young married woman died of phthisis some sixteen months after the birth of her first baby. Her husband within a year of his wife's death showed signs of the disease, and has been ailing more or less for eighteen months. There is no phthisis in his family, and he is of a strong constitution.

53.—In 1877 I attended a young man, aged 23, for phthisis; he died May 25, 1877. One of his parents, I believe his father, died of phthisis. His wife, a strong healthy girl of about 20, without any previous family history, as far as I could ascertain, of phthisis, commenced to be ill in the early part of 1880, and died of acute phthisis on March 10, 1880. The house they lived in was very small, ill-ventilated, and in a close, poor neighbourhood.

54.—Two cases: disease communicated to wives by husbands. No hereditary predisposition in either case. The parents were long-lived.

Supposed cause: anxiety, watching, and close attention to the husbands during their illness. The wives were exposed to bad hygienic conditions, and took no due precautionary measures, seldom breathing pure air.

*Case 1.*—This occurred in *my wife's sister*, who previous to marriage was robust and strong, was the mother of a son who is still living. She married a gentleman of delicate constitution, although no member of his family had died of phthisis. Had every comfort, and lived in a well-furnished house by the sea-side upon the eastern coast. For two years before his death he manifested lung disease. She was scarcely ever permitted to leave him, and for the last three months of his life she did not leave the house. For some months after his death she remained in perfect health, until an attack of bronchitis set in, after which the lungs became diseased, and she died suddenly from the giving way of an abscess.

All the surroundings, except the close attendance on the husband, were good. No hereditary disease in her family. Most of them long-lived. The father just 90 when he died. Her sisters and brother are in sound health, three being over 60.

*Case 2.*—A widow; age in 1879, 25. Two healthy children; a fine, handsome woman, of splendid physique. Never had any particular illness. Both parents were healthy, and without consumption on either side. Her first husband was ill for two years with phthisis, and she was his constant attendant, seldom quitting the room for days together. Slept in the same bed up to the day of his death. In August, 1879, she effected an insurance upon her life for the benefit of the children. No doubt at that time she was in perfect health. On Dec. 1, 1879, was attacked with pain in the left side, followed by most distressing cough, resulting in pulmonary mischief. In

February, 1880, she again married, went to the Isle of Wight, returned to Lee, Kent, where she died February 12, 1881, from general tuberculosis. Her surroundings were in every respect good.

55.—In 1877, T. W.'s wife came under my care for acute tuberculosis, of which she rapidly died.

Her family history was a consumptive one.

Shortly after her death, her husband came under my care for fistula in ano, from which he had suffered some eighteen months. The fistula was a blind internal, occasionally bursting; the sac of the abscess was very large. I operated, and in some six months' time succeeded in curing him. During my attendance I noticed extensive dulness and crepitation in the apex of the left lung, and other symptoms of phthisical disease, to wit, emaciation and night sweats. He was pronounced consumptive by a physician who examined him. He had always been a strong healthy man before marriage, and his family history was excellent. Under prolonged and careful treatment he was restored to health, the phthisical symptoms disappeared, he became strong and fat, and in time married again and became the father of healthy children.

Treatment extended over ten months.

1. External measures:—Flying blisters at intervals, painting with tr. iodi. of double the B. P. strength, inunction with croton oil, and tartrate of antimony ointment.

2. Internal measures:—Nux vomica, gentian, mineral acids and liquor strychnie for dyspepsia. Afterwards iron in various forms, quinine and phosphoric acid. Cod liver oil and Maltine also used when they could be borne.

3. Hygienic measures:—Rest, seaside, good light food, salt baths, friction of chest with strong brine, followed by sponging the chest and back with a saturated solution of ammonium chloride  $\frac{3}{4}$  ij, to which acetic acid and spirits of wine were added equally to make up a pint. Chest also rubbed with St. John Long's liniment.

56.—Yesterday, January 10, 1883, I saw a widow with consolidation of lung. History of occasionally slight hæmoptysis.

Her husband died of laryngeal phthisis about six months ago. For some time before his death, I advised her to sleep in another bed.

No cases of phthisis in widow's family nearer than grandfather and aunt. The father, mother, brothers, and sisters, of widow, were all free from any disease akin to phthisis.

Widow has all signs of phthisis.

Other cases which I cannot now recall.

57.—I have seen 21 instances, in twenty-two years' observation, all instances between married couples (six males, fifteen females) and all instances of profuse purulent expectoration of the communicant many months before death.

All are cases of (supposed) infection by deceased sufferers, which could not by the severest inspection be referred to heredity. That is to say, in questioning a widow whose husband had died of phthisis, if any evidence of the existence of phthisis among her blood relations is discovered, her case is immediately removed from the supposed infected list and classed among the hereditary.

No instances met with between sisters occupying the same bed. On the contrary.

In the family A., consisting in 1862 of two brothers and three sisters, three have died of phthisis during the past twenty years; two have had hæmoptysis and decided physical signs. Of the three sisters, the eldest has escaped, the second was in an apparently hopeless condition for more than a year (1872), but has since recovered; the youngest, who slept with the invalid sister, has shown no physical signs, but has had two attacks of acute rheumatism.

Of the family E., six in number, five have died of phthisis. Of two sisters with eighteen months difference of age, who slept together, one died at twenty, the other at forty-five years.

58.—April, 1882. Husband and wife. History of wife's family good, I believe.

59.—I have observed communication in the advanced stage. One very marked case in 1877, from wife to husband, husband's family free from phthisical tendency.

60.—A beerhouse keeper, aged 36, caught cold and died of phthisis within six months. His wife also died of phthisis after a lingering illness thirty months afterwards. I cannot trace any family history of phthisis.

The case came under my notice ten years ago.

61.—One case in particular where the wife, aged 30, died of phthisis, and the husband shortly after manifested symptoms of the same disease. He was ill for three years, and then recovered sufficiently to resume his occupation, that of a lightsman.

The wife's family was consumptive, but not the husband's.

Date, as nearly as I can remember, ten years ago.

62.—Certainly three or four cases.

Have a case now. Husband taken it from his wife. No family predisposition.

63.—August, 1878. Communicated to wife, Mrs. R., who nursed her husband during his last illness.

With the exception of a brother who was said to have died of decline, the family history of the wife was remarkably good. Both parents and grandparents lived to an advanced age, and, as far as is known, no other relatives have shown signs of phthisis.

Mrs. R. before marriage was strong and robust.

64. I have seen several cases.

March, 1880. The patient was quite healthy himself, and his family history was good, until his marriage a year before. His wife had a family history of phthisis, and died from the same disease. He began to show symptoms about three months before her death, and has a large tubercular deposit in the right lung now.

65.—Wife to husband.

Wife's father and mother healthy, died 75 and 82. Her father's family, that is, his brothers and brother's children, phthisical. Her mother's not so.

Husband, to whom communicated, previously healthy, and belonging to a healthy and non-phthisical family.

Husband ill at time of wife's death, and died within following year.

Large family, five or six twins. Two eldest have died of phthisis at about 20. Others at present, I believe, healthy, but none have reached the age of 20.

66.—One case. Madeira, April, 1866. Husband and wife.

Husband died of phthisis, hereditary, mother's side. Wife strong and healthy. No family history of phthisis.

Among the Portuguese and Spaniards it is a popular belief that phthisis is contagious. During illness a separate set of crockery, knives, forks, spoons, and bedclothes is put aside for their especial use. After death, bed and linen are destroyed.

67.—In 1874 a young woman died of phthisis, which appeared about a year after the death of her husband from the same disease. She had nursed him incessantly during the last weeks of his illness, and always slept in the same room with him. She never entirely regained strength after this, but no decided symptoms of tuberculosis showed themselves until after the interval stated.

There was no family history of the disease.

Patient was a female attendant in the Rainhill Asylum, Lancashire.

68.—A friend of my own has now a son in Algiers, believed to be in the last stage of phthisis. I cannot discover that any blood relative of his has been consumptive, but his wife died from consumption, and he was in constant attendance upon her during her illness.

69.—J. C. S., miller, aged 48, died December 27, 1882. Consumptive ten years, diarrhoea six months. His wife has now been suffering from consumption for three years, and is in the last stage. This woman was healthy when she married twelve years ago, has had five children, all healthy-looking, alive, and doing well. Her father is alive, her mother died about two years ago, aged 68. None of her



brothers or sisters have died of consumption, or are in consumption, though they look strumous.

This woman was in Brompton last year, and they made out her case to be one of contagion.

70.—Husband and wife. Husband died November, 1882, and was nursed by wife. One child died of tuberculosis. No family predisposition on either side.

NOTE.—Physical signs first noticed in wife in beginning of December, 1882. Husband aged 31, wife aged 28. She nursed him, slept with him, and took the sole charge of him. They kept a beerhouse, which is very damp. Wife still alive (January 3, 1883). Course of husband's disease acute.

71.—Mrs. G. T., 38, quite healthy till last Christmas, is now under treatment for phthisis.

Her father was healthy, died at 73; her mother was healthy, died at 62.

She has three sisters, aged 44, 36, and 34, and two brothers, aged 48 and 30, all healthy.

She married (1) sixteen years ago. First husband died seven years later, aged 40, supposed to be of phthisis. Knows nothing of any member of his family. By him she had three children, the first stillborn, the second died two hours after birth, the third still living, 13 years old, healthy.

(2) Three years ago she married C. T. His father died at age of 93. Mother died when he was 3 years old, disease unknown. Three sisters died of consumption, ages not recorded. A brother also died, disease unknown.

This second husband had been quite healthy until six months ago, when he went under treatment for cough, loss of flesh, and weakness. He was completely confined to his bed from the middle of January last till he died, February 3, 1883, of phthisis, in his 43rd year. She alone nursed him till the beginning of January, 1883.

72.—I can call to memory (I regret I have not notes or dates) at least five or six cases, where, without hereditary predisposition, the disease was unquestionably contracted through habitual intercourse with the affected. Two of these cases struck me at the time as most remarkable, and bearing directly on the communicability of the disease. Two women who constantly attended, and slept with their husbands up to a short time before the deaths of the latter, contracted the disease and died within twelve months of its inception. In neither of these cases was there any family predisposition for two generations.

73.—Husband to wife. W. S., aged 31 at death, first came under my observation early in 1879. His circumstances were very poor, so that he was nursed entirely by his wife. In September, 1879, he had a large cavity at apex of right lung, and he died January 23, 1880. Family history not known.

E. M. S., wife of above, was a strong, healthy-looking woman, about 26 at time of death, one of a family of six, having one sister and four brothers all living, and, with the exception of one brother, who was stated to be "delicate," but was quite able to do his work, in robust health. E. M. S. began to lose flesh from the time of her husband's death, being troubled by shortness of breath and cough. In August, 1881, there were the physical signs of a cavity in her right apex, and she died January 25, 1882.

74.—Maria B., aged 34, died January 29, 1883. Her first husband, to whom she was married, I believe, only ten months, died of consumption. She had no family history of phthisis in either parents, brothers, or sisters, according to what I have been told on inquiry. I was first called to see her June 29, 1877; she had not many weeks been re-married, and she was only a widow a few months.

75.—About twenty years since I had the misfortune to lose my wife, who died of phthisis. A few months afterwards I had severe cough, with blood-spitting and loss of flesh. I consulted Dr. —, one of the physicians of the Consumption Hospital, Brompton; and he said I had tubercles in the apex of my right lung, which I must, he said, have contracted from my wife. He ordered me to Australia, but, as I could not go there, I went to Pau, South France, where I spent a winter. I gained flesh,



lost my blood-spitting, and ever since have been able to carry on my professional duties, including night work. I am now in my 64th year.

**76.**—A., married at 19, B., aged 21, who was phthisical. No tendency to the disease exists in parents or grandparents of A. Her brother and sister are now healthy. She slept with B., had one child by him, nursed him through his disease till he died. She came home to her parents, having caught the disease, and died of phthisis in six months.

I look upon this disease as communicable from one person to another, from the frequency with which I have seen people who nursed, or came in close contact with, patients suffering with phthisis, afterwards dying of the disease themselves, when I knew of no history of it in themselves or their family.

**77.**—In 1862 a patient of mine, J. F., died of phthisis with cavities, hæmoptysis, &c. His wife nursed him very closely and carefully, sleeping with him to the last. About ten months later I found her lungs full of tubercles, and she died in six months of rapid consumption, also with cavities and hæmoptysis, sixteen months after her husband.

The three daughters, all the children she had, are all living in Truro at this moment and in good health.

The family on both husband's and wife's side was long-lived, and no consumption seems ever to have been known on either side.

**78.**—Mrs. L., of a phthisical family, died of phthisis in 1877, after an illness of two years.

Her husband, a commercial man, accustomed to live freely before his wife's death, and who became rather intemperate thereafter, developing enlarged liver with accompanying hæmatemesis, suffered from dry hard cough at and after her demise, till some six months ago, when he had profuse nocturnal perspirations and rapid extensive deposition of tubercle in the right lung. This quickly broke down, and terminated his existence on January 6, 1883.

He came from healthy parents. His mother died, over 60, of apoplexy two years ago. His father is living, about 70. Neither parent's relatives give tubercular histories. All his children, brothers, and sisters are living and healthy.

NOTE.—I have previously seen more than one case of husband following wife, or wife husband, of phthisis where the first had died of this disease. Unfortunately the only record I have is my memory. Still I have known many cases where one married partner having died of phthisis the survivor has not developed it, nor died of the same disease.

**79.**—I have seen four cases.

*Cases 1 and 2.* Two wives, with good family histories, were married in succession to a phthisical husband, and died with well-marked tuberculosis. Husband is also dead.

*Case 3.* A lady, whose family history, and health previously to marriage, were known to me to be good, contracted phthisis from her husband. The husband is now dead. She suffers occasionally from hæmoptysis.

*Case 4.* Wife contracted phthisis from husband. Husband dead. Wife in bad health, with signs of phthisis.

Family history of wife good.

**80.**—Mrs. L., a farmer's wife, whose father and sister had died of phthisis, consulted me in May, 1876. I attended her until September, 1876, and she died in Montgomeryshire of phthisis in December. In the following August (1877) the husband was seized with pneumonia, and ultimately died of phthisis the following December. Up to that time no member of his family for generations had been affected with it. I always regarded the taint as having been caught from his wife.

**81.**—April 23, 1879. Husband of wife, who died a month previously, had hæmoptysis on this day. The wife lingered eight months, and even in the last stages the man slept in the same bed in a small unventilated room. He also attended her almost

entirely by day until the last week or two, when the sister came to help. No one else in house.

He was a Crimean soldier, hardly ever ill in life. No consumption in his own family. But he had been a very free drinker, and consequently was a very dissipated character, until the last three years before above attack.

82.—One instance only, which made a great impression upon me at the time, and has made me ever since alive to the possible contagiousness of phthisis. This is now about eighteen years ago.

The wife, middle-aged, was suffering from advanced phthisis, when her husband began to lose flesh, had a constant cough, and his lungs on examination gave the physical signs of phthisis. He slept in the same bed as his wife, and complained of the smell of her breath.

His own family was quite exempt from the disease.

He finally recovered his health some months after his wife died.

83.—One case observed six months ago, and still under observation.

Husband died of phthisis. His wife, the present patient, nursed him, and up to a few weeks of his death slept with him.

Several other cases have been noted. I hope to present some of these in a tabular form soon.

The parents of this patient were not consumptive. A brother had a "weak chest."

84.—Husband died of phthisis in 1876. Wife a very healthy young woman, with no family history of phthisis, died two years after (1878) of phthisis.

No relationship.

85.—1882. Husband and wife. Patient's husband under my treatment for phthisis early in 1880. Died September 5, 1880. Patient slept in same room as her husband during his illness, became phthisical early in 1881, and is now dying.

Patient's father died at an advanced age from a "fit." Mother alive, aged 73. Grandfather died at 66; grandmother at 80; causes of death unknown. One brother died of phthisis, four alive. Two sisters dead, one of consumption, one in childbirth. One alive and well.

86.—Mrs. M. L., aged 26, after nursing her husband, who was suffering from phthisis, for about two years, came to me on the 8th of this month, a week or ten days after her husband's decease, to be examined. I found disease in the apex of each lung, the left being dullest on percussion, and on auscultation giving very distinct large moist rales. I could not get any direct or indirect paternal phthisical history, or maternal; but she had a sister who died of acute phthisis of three weeks' duration, whose age was 15.

87.—I have seen two cases.

Case 1. F. F., aged 25, farm labourer. Wife died two years ago of phthisis, since then has been wasting. I am attending him now. He is in the last stage of phthisis. His father and mother living and well. No relative on his side has died of phthisis. Date March, 1883.

Case 2. P. MeN., carpenter, aged 35, died of phthisis six months after his wife, who died of it also. All his family free of the disease. He slept with his wife to the last in a small close room. Date July, 1870.

88.—One case, February 14, 1883. Husband to wife. No relationship before marriage. Family predisposition on the husband's side, who has a sister phthisical. No hereditary taint on the wife's side traceable previous to marriage.

89.—Case in 1866. Husband and wife. Not any family predisposition.

90.—In the autumn of 1880 a man developed phthisis and died. His wife nursed him and occupied the same bed during his illness. She took ill and died of phthisis in the spring of 1881. No predisposition.

91.—I am attending a woman in the last stage of phthisis, who seems to have contracted the disease from her husband. There is no tendency to the disease on her side, as neither parents, brothers, nor sisters have suffered from it. She nursed her husband more or less closely for about three years. He died eight months ago, but she had shown symptoms several months before his death.

92.—*Case 1.* Observation made March 12, 1883. Widow aged 27. Has had four children, three living and healthy, youngest aged 12 months. Illness began after birth of last child. No case known in the family before. Father and mother and a brother and sister living. Husband died of pulmonary phthisis in August last. His family stated to be very consumptive.

*Case 2.* Widower. Not seen for some time. Case of hæmorrhagic phthisis. No family predisposition. Wife died from pulmonary phthisis. I believe this man has recovered, but further particulars can be obtained if desired.

93.—I particularly remember forty years ago the cases of my predecessor and his two wives, all of whom died from phthisis. The first wife died about the age of 38. Had three children. Her father and mother died aged 70 or thereabouts. The second wife married rather late in life, that is, about 44, and showed symptoms of phthisis a few years afterward. She survived her husband about eight years, he having died at 49, between the times of death of the two wives.

Subsequently, twenty years since, I attended a man and wife in good circumstances. The husband first took the disease, and died early; the wife a year or so later, and also a daughter aged 14.

Others I might give note of if time permitted.

94.—Mr. E. died of acute phthisis six years ago, after a very short illness, which was attended by profuse expectoration. Mrs. E. closely nursed her husband, and two months, she thinks, after his death she commenced to cough. About this time consolidation was found at the right apex, and there are now signs of phthisis over the upper half of the right lung. She has had frequent hæmoptysis since the commencement of her illness, and has never been free from cough and expectoration.

None of Mrs. E.'s relatives have had phthisis, and her three children are healthy. Mr. E.'s father and two of Mr. E.'s brothers died of phthisis.

95.—February, 1882. Wife from husband. All wife's family healthy so far as could be traced. Father and brother on husband's side consumptive. Husband died a few months after marriage. Wife came under my care two months afterwards.

Husband from wife. His family all healthy, and a healthy man himself. Wife's family: father and two brothers died consumptive. Were married two years when wife died of same. Husband began to show symptoms immediately after, and died in six months. Had two children, both strumous.

96.—1873 to 1879. Husband and wife.

Wife: Fair, delicate, and advanced in consumption in winter of 1873. Family history of phthisis. Died early in 1877. Resided in West Indies between years 1873 and 1876. Died in England.

Husband: Dark complexion, robust, athletic, active, and sound in 1873 and to 1876. No trace of phthisis during that period. In autumn, 1877, husband came under my care in England for phthisis, and died in 1879. Spent winter abroad. Died of phthisis and pneumonia. Had primary syphilis before marriage, and appeared to have recovered, but first child had congenital syphilis and died. Second child died of bronchitis. Had syphilis again after wife's death, and in my opinion syphilis called on or intensified the phthisis.

97.—*Case 1.* In 1880. From husband to wife. The husband dying in 1881, the wife in 1882. I know of no predisposition to the disease on either side.

*Case 2.* In 1882. From wife to husband. Husband died of acute tuberculosis. A sister of his died in the same year from chronic phthisis. Wife still alive, ill four years. Her mother died in 1882 of phthisis.

98.—Some years ago I attended a working man's wife suffering from phthisis.



The man was remarkably healthy and robust. He paid her a great deal of attention. They lived in a very small cottage. About the time of the wife's death the husband was seized with hæmoptysis, and died in a few months of phthisis. I can obtain no information of family history. A most clear case of contagion I thought.

99.—The wife, aged about 48, was perfectly well eight months before I visited her. No family history of phthisis. Apices dull, tubular breathing with crepitations. Her husband died of phthisis eight months before, and during his illness she nursed him. She also slept in the same bed, and to this she attributed her complaint. She was a dispensary patient (under my care, I believe, for three months, at the end of which time she died), therefore not in good circumstances.

100.—Observation made in April, 1876. Communicated from husband to wife. Latter nursed former almost constantly in small, overheated, badly-ventilated apartment. No family history of phthisis in wife's case, but in April, 1875, she suffered from a normal attack of pleurisy, from which she made a good recovery. This probably predisposed her to phthisis. She became ill soon after husband's death, and lived only six or seven weeks.

101.—Three distinct cases, some ten to fifteen years since, where wives apparently contracted phthisis from husbands who died of that disease. In each case the wife had a good family history, and free of tubercular disease. No blood relation existed between the parties.

## GROUP A.

## Section II.

NAME AND ADDRESS OF OBSERVER.	NO. OF RETURN.
Mantle, Alfred, M.D., West Stanley, Durham . . . . .	102
Smith, R. Shingleton, M.D., Clifton . . . . .	103
Thorowgood, John O., M.D., 61, Welbeck-street, W. . . . .	104
Thorp, Charles W., M.R.C.S., Todmorden . . . . .	105
Mackie, W. J., L.K.Q.C.P.I., Turvey . . . . .	106
Heath, W. Lenton, M.B., F.R.C.S., 85, Glo'ster-road, S. Kensington, W. . . . .	107
Gourney, B., M.R.C.S., Salisbury . . . . .	108
McVeagh, John, M.D., 1, Rutland-square East, Dublin . . . . .	109
Cameron, J. Spottiswoode, M.D., Huddersfield . . . . .	110
Harris, J. Delpratt, M.R.C.S., Exeter . . . . .	111
Perry, Robert, M.D., Glasgow . . . . .	112
Bowen, O., M.R.C.S., Liverpool . . . . .	113
Smith, Samuel W., M.D., Pershore . . . . .	114
Robinson, Frederick, M.D., F.R.C.P., Eastbourne . . . . .	115
Prowse, William, M.R.C.S., Cambridge . . . . .	116
Price, Edwin, M.R.C.S., Dudley Port, Tipton . . . . .	117
Jackson, George, F.R.C.S., Plymouth . . . . .	118
Smith, Joseph Evans, M.R.C.S., Snodland, Rochester . . . . .	119
(Return with no name) . . . . .	120
Smith, Samuel, M.R.C.S., Carlton Colville, Lowestoft . . . . .	121
Barrett, Alfred E., M.R.C.S., 12, Ladbroke-grove, W. . . . .	122
Denne, Henry, M.D., L.R.C.P., 8, Hagley-road, Edgbaston, Birmingham . . . . .	123
Hardwicke, Junius, M.D., F.R.C.S., Rotherham . . . . .	124
Boyce, Charles, M.B., Maidstone . . . . .	125
Russell, A. J., M.D., Denver, Colorado, U.S. . . . .	126

NAME AND ADDRESS OF OBSERVER.	NO. OF RETURN.
Prinee, C. E., M.R.C.S., Buekhurst-hill . . . . .	127
Britton, Thomas, M.D., Halifax . . . . .	128
Elliott, Ernest, M.D., Purbrook, Cosham . . . . .	129
Clapperton, James, L.R.C.P., Broughton, Winchester . . . . .	130
Solomon, Jas. Vose, F.R.C.S., Birmingham . . . . .	131
Loché, Thomas W. S., M.R.C.S., Coatham, Redcar . . . . .	132
Payne, Henry, M.R.C.S., Loxley, Sheffield . . . . .	133
Uthoff, John C., M.D., Brighton . . . . .	134
Purdon, H. S., M.D., Belfast . . . . .	135
Darwin, E. H., M.D., M.R.C.P.E., Albert Park, Didsbury . . . . .	136
Whittle, Ewing, M.D., Liverpool . . . . .	137
Whiteley, John, M.R.C.S., Sonthgate, Wakefield . . . . .	138
Rooke, J. Morley, M.D., Bays Hill, Cheltenham . . . . .	139
Frazer, R. T., L.K.Q.C.P., Lavender Hill, S.W. . . . .	140
Harper, Joseph, L.R.C.P., Barnstaple . . . . .	141
Alderton, Frederick H., M.D., Hammersmith, W. . . . .	142
Ransford, T. D., F.R.C.S., Bath . . . . .	143
Bayfield, Horace O., L.R.C.P., Lavender Hill, S.W. . . . .	144
Kilburn, William B., M.R.C.S., West Aneklund, Bishops Auckland, co. Durham . . . . .	145
Gill, George, L.R.C.P., Liverpool . . . . .	146
Kenyon, John E., L.R.C.P., Bradford . . . . .	147
Flint, Arthur, L.R.C.P., Westgate-on-Sea . . . . .	148
Meadows, Henry, M.B., Leicester . . . . .	149
Hoyle, William, M.R.C.S., Tyldesley, Manchester . . . . .	150
Hinton, Joseph, M.R.C.S., Warminster . . . . .	151
Emmerson, William L., L.M.Durh., Waltham, Melton Mowbray . . . . .	152
Edmunds, James, M.D., M.R.C.P., 8, Grafton-street, W. . . . .	153
Clibborn, William, M.D., Birmingham . . . . .	154
Wolfenden, J. W., L.R.C.P., Tntbnry, Burton . . . . .	155
Jones, Evans, M.R.C.S., Ty-mawr, Aberdare, S. Wales . . . . .	156
Ferris, John Spencer, M.B., Uxbridge . . . . .	157
Hickes, Thomas, M.R.C.S., Cheddar, Weston-super-Mare . . . . .	158

102.—August, 1882. Husband and wife. M. S., aged 31, a miner. Always enjoyed good health until spring of 1881, when he noticed a small swelling in right axilla, which grew in the same year as large as a good sized apple, under the pectoral muscle. He was treated by a quack in Durham with plasters, which caused great suppuration and pain. His wife was lying in bed with phthisis far advanced, with whom he slept and lived in a small close room. On examining his chest, I found signs of acute phthisis. He died in six weeks, his wife living five months after his death. The tumour I believed to be a lymphoma. There was no history whatever of phthisis in his family, nor could any consumption be traced in the family of his wife. The disease was of two years' duration in her case, and she was 25 when the first signs were noticed.

103.—*Case 1.* Husband and wife living in village near Bristol. Both died within a day of each other of tubercular phthisis six years after marriage. The wife, aged 30 at death, August, 1882, was one of five healthy sisters, whose father died of consumption in 1871. Three years after marriage the husband, aged 26, whose family history was free from consumption, got a succession of "colds," and was never well afterwards. Two years after the commencement of the husband's illness, the wife "caught cold," spat blood, and then failed in every respect. She was in constant attendance on, and cohabited with, her husband during the whole period of his illness.

*Case 2.*—A young married man, whose wife, much older than himself, had recently died of phthisis. His family history was good, father and mother both

living over 60, and no history of phthisis in other members. His own health had been good till six months after the death of the wife, when he noticed a little cough and expectoration in the mornings, with occasional streaks of blood. Hæmoptysis in small quantities occurred in October, 1880, November, December, twice in February, 1881, and in March there was erepitation at the left supra-scapular region. In March, 1882, patient had quite recovered. There were no physical signs of disease, but he was seldom free from colds, and had lost more than a stone in weight.

104.—Some years ago, a woman at Vieteria Park Hospital. I examined the lung apices, and found no sign of disease, but at the right base we all thought there were cavernous sounds. All the other parts of the lungs were sound, and her general health had been good. On inquiry I learned that she had been in close attendance on her husband, who had died of phthisis. I heard some months after that the cavity seemed to progress.

This is all my experience in evidence of contagiousness of phthisis.

105.—I have observed many cases where I have felt convinced the disease has been communicated. The number I cannot give. It has been chiefly from wife to husband, and *vice versa*. In some of the cases there was a family predisposition.

106.—Observation made in 1878. The disease appeared to be communicated from wife to husband, developing after her death. The parents of husband were free from the disease, and lived to old age. A brother of the patient suffered from the disease.

107.—One case from wife to husband in 1879. Family predisposition to the disease on the part of the wife (one sister died of phthisis). None on part of husband. Husband first showed signs of the disease when it had far advanced in the wife.

108.—Have known several instances where a wife, healthy at the time, has nursed a phthisical husband, and has herself developed phthisis within three years from the date of her husband's death, and died. Have no record of the family history in the cases. Observed within the past ten years.

109.—Two cases in note-book, occurring between husband and wife. No hereditary tendency to disease from either parent.

Case 1.—1857.—Husband died of phthisis, Co. Wicklow. Wife 4 years afterwards in England.

Case 2.—Husband died of phthisis. Wife now in chronic phthisis, evidently communicated from husband.

110.—About two years ago an out-patient of mine at the Huddersfield Infirmary of, as far as I remember, healthy family history. She had apparently contracted the disease from her husband.

111.—I have observed two cases. In each case the husband was the first attacked.

Case 1. The first case, a carpenter, sent from Yorkshire to Torquay, had all the symptoms and physical signs of phthisis. Wife well up to time of his illness, but soon after he took it she became just like him. Both at present survive.

Case 2. In the second case the wife's parents were living, and she denied having the complaint in the family, and was most certain in her own mind, that her husband, who had just died when I saw her, had given it to her, being perfectly well up to the time he was taken. The result was death.

112.—During the past twenty-five years I have seen numerous instances, particularly in the case of husband and wife, when without any hereditary or family predisposition the disease has been communicated from one to the other, more frequently from husband to wife.

113.—I have seen a case from husband to wife, but took no note. I cannot give the family predisposition, but as far as I remember, there was none on the wife's side.



114.—Have seen several cases; E. G. P., lost his first wife of phthisis, remarried and himself died of phthisis; subsequently his second wife (and widow) died at 35 of phthisis. I knew her as a strong buxom woman for years.

115.—Notably in the case of a young surgeon whose wife, constantly ministering to him (his case was chronic phthisis), became soon after his decease affected with the ailment and died. She was of healthy physique, as were her relatives.

116.—My own brother, a surgeon in practice at Clifton, was married three times. The two first wives died of phthisis. His third wife still survives him, he having died after prolonged suffering from the same disease. Tubercular disease is unknown to any members of his own family. He died at the age of 49 in the year 1864. He lingered for seven years.

117.—Suspected it in two cases.

118.—Have certainly seen one case of communication from wife to husband, and suspected it in another.

119.—In the year 1874, I had two cases of consumption at Hay, Breconshire, a married couple, Mr. and Mrs. H. I was first called to the husband, he died of hæmoptysis after six months. His death was hastened by intemperate habits. His wife had suffered at the same time for months from cough. After his death I attended her, and she died in about two months after her husband with manifest symptoms of phthisis. I believe that the husband caught the disease from his wife, as there was no history of phthisis in his family, and there was in the wife's. Father and sister, if I remember right.

120.—Several years since I saw two or three cases, but do not remember the particulars, excepting the fact that the disease appeared to be transmitted from husband to wife, and *vice versa*.

121.—I am of opinion that consumption is communicable from one person to another, and have attended cases in which I have reason to believe it was so. Having kept no record, I cannot give details or particulars. One was of husband and wife.

122.—From 1866 to 1872 I attended Mrs. K., aged about 30, who died of phthisis in January, 1872. Her husband, with no phthisical history, developed laryngeal phthisis, and died in April, 1872. Living in a country district in Norfolk. Have observed other cases, but have no record.

123.—I find a record of thirty-one fatal cases of phthisis during the last thirteen years. In only one case is there any reasonable probability of its having been communicated. It is that of a male who died in 1881, aged 35. His wife died with phthisis of three years' duration in June, 1879. He continued apparently healthy until September, 1880, when hæmoptysis occurred, and other phthisical conditions followed.

His parents and grandparents, brothers and sisters, are not phthisical, but his mother's brother died of hæmorrhagic phthisis, aged 23.

124.—Many cases from husband to wife. From recollection I believe the family history was good.

125.—I have observed lately one decided case, but I know of three others less marked; in each case the disease was communicated from husband to wife, or *vice versa*. In one case there was family predisposition.

126.—I have no records, but am satisfied in my own mind that I have observed cases which I regarded at the time as having been contracted by close contact in case of husband and wife.

127.—I have seen several instances. Date from 1860 to 1870. Husbands and wives.

128.—I think I have seen such cases. From husband to wife, and *vice versa*. Cases not recorded, but very few. Only two or three satisfactorily so accounted for.

129. I have frequently seen cases, and I read a paper on this subject, I think in 1856, before the Portsmouth Medical Society, which was published in the *Medical Gazette*.

130.—Mrs. C. died last year from phthisis. Her husband is now suffering from the same disease. He belongs to a healthy family. I believe there were three other cases of contagious pulmonary phthisis which occurred in my practice, but have no notes of the cases. They were wives and mothers of families.

131.—I have seen such cases when a general practitioner, and was in the habit of cautioning wives against sleeping with their husbands who were in the advanced stages of pulmonary phthisis. My conviction was attained by inability to trace hereditary taint in the wife, who had nursed her husband. I regret that I have not preserved notes.

132.—During twenty-five years' practice I have had cases of deaths from phthisis in married people, and either husband or wife had no trace whatever of family predisposition.

133.—I have seen many cases from husband to wife, and wife to husband, during the thirty-nine years I have been in practice.

134.—L. T., female, age about 50, died of phthisis and tubercular meningitis April, 1882. No phthisis in family. Husband died of phthisis some years (ten?) before. About a year or two afterwards she developed distinct chest affections. These particulars are only given from memory.

135.—In 1870. Patients, man and wife. Under observation for about one year. No history of phthisis in either families.

136.—I have seen several cases. Last case, Mary W., aged 31. Husband died of phthisis, and she previously healthy, and from a healthy family, contracted the disease and died of it during 1881.

137.—I have during forty years' experience met in a few instances with such cases, chiefly between wife and husband, so that for many years I have felt certain that the disease is communicable when the surroundings are much the reverse of sanitary.

138.—During a practice of twenty years I have seen, I think I may say, from ten to twenty cases where it has been communicated from husband to wife, and *vice versa*, without any, as far as I could glean, hereditary predisposition. When the same bed has been occupied during the last stage of the disease, the transmission has more frequently occurred and the development has been more rapid.

139.—Four cases, three from the husband to the wife, and one from the wife to the husband. One case is specially noteworthy, in that the husband was affected with chronic phthisis for which I attended him. His wife was a strong-looking woman. They went abroad. He still lives, but his wife fell into consumption and died.

140.—A case seen 3rd January. Died next day. A man said to have acquired phthisis from his first wife. His family quite free from the disease.

141.—In 1878. Husband and wife.

142.—I have seen several cases, one where a first wife gave it to husband. He was very healthy when married, and remained so until about a year before his first wife's death. He then had slight dulness at apices, cough, and a little bronchitis. After her death he had three distinct attacks of hæmoptysis, with consolidation of both lungs. Subsequently married a healthy wife, became comparatively healthy, but have lost sight of him. There was no history of phthisis on his side.

143.—One case observed from September, 1878 to middle of 1880. Wife observed to have phthisis of apex of one lung, which rapidly progressed. Then the husband, a joiner, caught a cold at his work which never left him and developed into phthisis. I fancy there was a predisposition on husband's part.

144.—I have seen at least twenty cases, most frequently between married people who sleep together. One being phthisical, the other contracts the disease.

145.—(1.) Husband and wife were both ill together, although the husband commenced first by two or three months. They both were ill for several months and died within a day or two of one another. Since then their daughter died of phthisis.

(2.) I also remember another case where husband and wife were ill at the same time. The wife began first and the husband followed a few months afterwards. They died within a week of each other.

146.—I have observed a few cases. One especially, when the husband appeared to be in the last stage. His wife took the disease and died in a very short time. The husband recovered sufficiently to resume his usual occupation for some time. No family history.

147.—One case particularly, where the wife attended to and waited on her husband most devotedly while on his death-bed with phthisis. A few weeks afterwards she commenced with acute consumption of the lungs and sank very rapidly.

148.—A patient of mine died of phthisis two years ago. His wife, who was quite healthy up to his death has during the last nine months developed the disease, which is now fully established. Am unable to give any history of family predisposition.

149.—I remember one case very well, but it was some years ago; a woman seemed to have been infected from her husband. I have seen many suspicious cases in my practice. In this district the public generally believe in its communicability.

150.—I have seen many instances, but most frequently between husband and wife. I have this year a man suffering from laryngeal phthisis, whose wife died last April from the same disease.

151.—Cases reported (1) from wife to husband, (2) from husband to wife; but family history not ascertained in one case, and apparently there was predisposition in the other.

152.—One case, about eight years ago, in which the wife died about four months after the husband, both of phthisis. I cannot give any family history.

153.—I have held for the last twenty years that tubercular phthisis is infective, as from husband to wife when sleeping together in ill-aired rooms.

154.—In 1881. Husband and wife. Parents healthy. Wife's sister died of phthisis. Wife died last November of phthisis. Husband, both lungs affected.

155.—Case in 1880. Husband and wife. Hereditary in husband.

156.—I have been several times struck with the frequency of phthisis in husbands and wives, but have kept no records of the cases.

157.—Several by husband to wife, and one or two by wife to husband.

158.—I have seen four or more cases.



## GROUP B.

*Returns of cases observed between members of the same family, between parents and children, brothers and sisters, as well as between husband and wife.*

## Section 1.\*

NAME AND ADDRESS OF OBSERVER.	NO. OF RETURN.
Robson, A. W. May, F.R.C.S., Leeds . . . . .	159
Duguid, W. R., M.D., Buckie, Banffs . . . . .	160
Kirkland, Robert, M.B., Cheltenham . . . . .	161
Rainbird, Horace, L.R.C.P., Saxilby, Lincoln . . . . .	162
Scott, E. S., M.B., Shrewsbury . . . . .	163
Dale, Ridley, L.R.C.P., Sunderland . . . . .	164
Thompson, Harry Y., M.B., Bramptou, Cumberland . . . . .	165
Dewau, James S., M.D., Arbroath, N.B. . . . .	166
Kelman, George, L.R.C.P., Gifford, Haddington . . . . .	167
Johnston, J., M.D., Bolton . . . . .	168
Fielding, Thomas, M.D., Milton Abbas, Blandford . . . . .	169
Fulford, W. E., M.R.C.P.E., Wadebridge . . . . .	170
Leadman, Alex. D. H., L.R.C.P., Boroughbridge . . . . .	171
Macphail, Donald, M.D., Whifflet, N.B. . . . .	172
Cunningham, John, M.B., Campbeltown, N.B. . . . .	173
Booth, J. Mackenzie, M.D., Aberdeen . . . . .	174
Rowe, William Palmer, L.R.C.P., Liverpool . . . . .	175
Kirk, Robert, M.D., Partick, Glasgow . . . . .	176
Evans, Thomas, M.D., Newquay, Cardigan . . . . .	177
Crawford, George, M.D., Port Glasgow . . . . .	178
Grayson, F. Dorrell, M.R.C.S., Rayleigh . . . . .	179
Drew, John, M.D., Stirling, N.B. . . . .	180
Newton, Lancelot, M.R.C.S., Alconbury Hill, Huntingdou . . . . .	181
Boyce, J. Wallace, M.B., Stillorgan . . . . .	182
Reid, James, F.R.C.S., Canterbury . . . . .	183
Ostlere, Robert, M.B., 47, Stoke Newington-road, N. . . . .	184
Blair, John, M.D., Shotts, N.B. . . . .	185
James, J. Brindley, M.R.C.S., Brindley House, Jamaica-road, S.E. . . . .	186
Kempe, Arthur, M.R.C.P., St. Sidwells, Exeter . . . . .	187
Ross, Roderick, L.R.C.P., Lochs, Stornoway . . . . .	188
Clarke, William Mitchell, M.R.C.S., Clifton . . . . .	189
Peart, Robert S., M.D., North Shields . . . . .	190
Budd, Christian, M.B., North Tawton . . . . .	191
Winckworth, Charles Edward, L.R.C.P., Shefford . . . . .	192
Bampton, A. H., M.D., Plymouth . . . . .	193
Sutherland, W., L.R.C.P., Newcastle-on-Tyne . . . . .	194
Stewart, Alexander, M.D., Pendleton, Manchester . . . . .	195
Dickinson, Thomas, M.R.C.S., 33, Sloane-street, S.W. . . . .	196
Satchell, W. A., F.R.C.P.E., Bournemouth . . . . .	197
Roberts, Carr Holstock, L.R.C.P., Herries-street, Harrow-road . . . . .	198
Davis, Thomas, M.D., Londonderry . . . . .	199
Brown, George Arthur, M.R.C.S., Tredegar . . . . .	200
Berry, F. C., M.D., Lynton . . . . .	201
Ellerton, Frederick C. Grant, L.R.C.P., Leamington . . . . .	202
Kavanagh, P., M.D., 186, Lewisham High-road, S.E. . . . .	203
Mellis, John, M.R.C.S., Fraserburgh . . . . .	204

\* In two sections like Group (A)

159.—I have observed a number of cases—*e.g.*,

1. No family history of phthisis in father and mother, *æt.* 50 and 52, strong and well now (1883).

A son, 19, chronic phthisis, died November, 1881. Nursed by sister, who was in robust health.

2. One month after his death she became ill, and died of acute phthisis in six weeks. February, 1882.

3. Within the month a son, 14, began to be feverish. Cough supervened, and he died of acute phthisis in April, 1882.

The family of several children left the house, since they thought it to be infected, and have had no other case.

160.—A woman died of the disease in August, 1880. Nursed by a sister, who consulted me for the first symptoms of the disease six or eight months after, and died of it in June, 1882. The two had occupied the same bed for some time until warned of the danger. Both parents lived to extreme old age.

161.—Last July I attended a young girl suffering from phthisis. On October 5 she died. About a week or so before her death a brother, who was much attached to her, spent all his leisure time with her. The brother was a strong, well-developed young man. In November he began to feel out of sorts, and a slight cough has troubled him since the middle of December. At present mucous *râles*, with prolonged expiration and slight dulness, can be detected over the middle of the right lung behind. He sweats much at night.

The father and mother are both living. There is no history of phthisis in the grandparents. The rest of the family are all quite healthy, and no death has occurred from phthisis but this.

162.—A patient now under treatment went to nurse her sister, who was suffering from phthisis in June, 1882. She appeared to be perfectly well when she left her home. A few weeks afterwards she was seized precisely in the same way as her sister, and is now in an advanced stage of phthisis.

No consumption on father's or mother's side.

163.—In January, 1880, M. T., the wife of a country postmaster, had been ill two or three years with phthisis. About the time the symptoms first showed themselves her stepson, aged 20, died of galloping consumption, and she had been his constant attendant and nurse. Previous to this she had always been healthy. No history of phthisis on either her father's or mother's side. Her youngest child, a little boy of 3, who was with her constantly and slept in the same room, died five weeks after her of the same complaint.

The mother of the stepson mentioned above died of phthisis when he was a few years old.

164.—In 1879 a husband died after six months' illness, during which time his wife slept with him and attended to him. The man, his wife, and six children, all occupied the same room. Shortly before his death the woman, who had previously been strong and healthy, and in whose history there was no predisposition to phthisis, presented symptoms of this disease, and a cavity formed in the left lung at apex a few weeks after her husband's death. Under treatment she improved, and in nine months was convalescent. The treatment consisted of iodine painted externally, and creosote administered by the mouth and by inhalation. Since then two children have died of phthisis.

165.—*Case 1.*—(i.) Elizabeth H., *æt.* 40. Phthisis 14 months. Diarrhœa 6 days. Died April 22, 1876.

(ii.) Mary Ann H., *æt.* 19. Eldest daughter of above. Phthisis 18 months. Hæmoptysis on three occasions. Died May 12, 1877.

(iii.) William H., *æt.* 12. Son of Eliz. H. Phthisis 6 months. Ascites 3 weeks. Died August 6, 1877.

(iv.) Another sister died within 18 months of the last case from phthisis, but was not attended by me.

No previous history of phthisis in families of parents or grandparents. The husband of Eliz. H. died from the effects of an accident.

All lived in one house.

*Case 2.*—(i.) Sarah A., *æt.* 42. Phthisis 4 or 5 months, which developed immediately after childbirth. Died April 21, 1882.

(ii.) John A., *æt.* 45 (?), husband of above, showed symptoms very shortly after his wife commenced her illness, and has now chronic phthisis with profuse expectoration, sweating, cough, diarrhoea, and loss of weight. He was formerly a strong healthy man, but always more or less intemperate.

No family history of phthisis on either side.

166.—At intervals during the last fifteen years have observed the following cases :

*Cases 1 and 2.*—Wife after husband twice.

*Case 3.*—Mother after 2 daughters.

*Case 4.*—Mother after husband, son and daughter.

*Case 5.*—Father after son.

In all the above cases the patients lived in small confined houses, and slept in the "box beds" in use in Scotland. During twenty-five years I have not seen one case of contagion in the airy houses of the well-to-do.

In the five cases above noted there was no history of family tendency to phthisis.

167.—An aunt of mine married a gentleman and had two daughters. He died of fever. She after a few years married another gentleman, and had two daughters. He died of phthisis affecting the larynx. She nursed and slept with him. Shortly after his death it was discovered that she had the disease, and she died in about five years. During this time her eldest daughter by the second husband, who also attended to and slept with her, became affected, and died a year after her. After the death of the mother, the eldest daughter of her first husband managed the house, and also slept with her sister till her death. She is now fast declining of phthisis.

The family history of the wife and first husband is very good. In no branches of the families had phthisis occurred.

I consider that it was communicated by the second husband to the wife, and by the third daughter to the eldest.

168.—One case in which there was no family history of phthisis. First a son died of it at about 16. A few months afterwards a younger sister succumbed to same disease, and she was followed in six months by the mother, and by the father in twelve months after the mother. Family well-to-do, but they displayed great ignorance of ventilation. Children slept in attic with an immovable skylight, and no fire-place or other means of ventilation. So morbid was the fear of draughts that the windows were hardly ever opened, and the atmosphere inside the whole house was most oppressive and stifling.

169.—In a family named C. it appeared that when the disease was introduced it attacked the members in succession. They were constitutionally very delicate. Family history not known. The mother died in childhood.

The daughter Leah died 1867. The son John 1868. Father 1869 or 1870. About the same time, son William, living away, married and paid a visit home. Shortly afterwards returned home and died. Son H. died in 1872. All from acute phthisis. There is a daughter left who was very delicate for some time, but, under good conditions, has survived. There was no doubt overcrowding in this house, but with the exception of this family, the village has been less afflicted with phthisis than any in my district. The cottage has since been occupied by elderly people.

A family named D—e had a similar history, but I cannot give particulars.

170.—In 1881 I attended a girl, aged 18, with vomica at apex of both lungs. She was occupying the same bed as her mother, aged 59, bedridden until her, the girl's death, August 16, 1881. The mother, M. O., complained of cough, July 11, for the first time, and during August had several attacks of hæmoptysis, and died from phthisis September 25.

There is no history of phthisis in any of the surviving members of the family.



171.—Mother from daughter.

A. F., aged 17, died October 21, 1881, from phthisis; Mary E. F., aged 36, died April 12, 1882. She had disease of the knee-joint of a chronic nature, and in spite of it attended to her daughter, who was ill about a year. She slept in the same bed the last few weeks of her daughter's illness, and directly after her daughter's death commenced to cough, the disease developed with great rapidity, and she died about six months afterwards from phthisis.

Family predisposition I cannot now remember.

172.—A. W., of a very healthy family, aged 20, died January 8, 1883, of phthisis, of one year's duration. Parents both alive and very healthy. Family history traced out several degrees gave no indication of phthisical tendency.

An elder brother died of rapid phthisis some months ago. He had been a healthy youth, but caught repeated severe colds from travelling by rail after violent exercise at football without changing his clothes. The course of that case was rapid.

A. W. slept with his brother in a concealed bed in a badly ventilated room for several months after the brother took ill. Previously he had been healthy, but not lusty. Health declined gradually, phthisis developed rapidly.

Other brothers and sisters are younger, and strong and healthy.

173.—(1.) Husband dies of phthisis in spring of 1879. Wife nurses him, begins to cough in April, 1879, dies in June, 1880, of phthisis. The wife was not related to her husband. There was no lung disease in any of her relations, near or remote. She belonged to a long-lived family. Her father and mother are still alive and well. Her grandparents died advanced in years.

(2.) A young man comes here in 1877 with fistula *in ano* and dulness in the apices of the lungs. Dies in October, 1878, of phthisis. Sister nursed him, begins to cough in 1878, October or November, removes to one of the Western Hebrides, continues to cough, returns here, dies in September, 1880, of phthisis. Only other child at home takes a cold in August, 1880, continues to cough, and dies in November, 1882, of phthisis.

Other members of the family are older than the above, and in good health. The father and mother are of families noted for longevity. Grandparents on both sides lived to extreme old age.

174.—November, 1882. Sisters. No history of family predisposition.

One sister died of phthisis, and the second, who had been previously quite healthy, for some time before her death slept in the same bed with her.

I have had several similar cases, where, either the husband, or wife, or a friend, who had slept with a phthisical patient, afterwards contracted phthisis, and that without a family history of the disease.

175.—Mrs. A., aged 48, died on January 8, 1883, of phthisis, suddenly contracted while nursing her son suffering from a similar disease. Six months ago he returned home from Africa, and she nursed him constantly and assiduously. She was then seized with acute phthisis and rapid breaking down of the apices of both lungs. Previously she had been a strong, robust, and vigorous woman, well nourished, and never ill.

No family predisposition to the disease among any of the relatives.

176.—One case under observation at present. The wife of a patient who has been the subject of slow but undoubted phthisis for two, or probably more years, and has been away from home four months. About two months ago she complained of severe cough, &c., and phthisis is now pursuing an extremely rapid course.

She never knew a case of consumption among her near relatives.

I can recall four other cases in which two or more brothers and sisters suffered from phthisis in rapid succession, the second falling ill before the first died, under circumstances which favour the supposition of communication from one to another having taken place.

One case was that of two brothers, who slept together, and who died within a few months of each other. Two sisters in the house remained well. In two other cases the houses were very badly ventilated.

177.—(1.) On March 1, 1879, I saw a young man, aged 20, in the last stage of pulmonary phthisis. He died March 26. It was alleged that he used to sleep with the son of another family, who had died of consumption a year before.

(2.) On March 1, 1879, I also saw the mother of No. 1, suffering from pulmonary consumption. It was said that her illness commenced after her son came home sick. She died July 26, 1880.

(3.) On March 27, 1879, I saw brother of No. 1 affected with chronic pleurisy (tubercular). He frequently slept with his sick brother after November, 1878. He died of tubercular meningitis, July 28, 1879. There has been no marked predisposition to phthisis in this family, and the father and several other children have remained healthy in the same circumstances since above date.

178.—A. H., aged 47, occupied a house of two apartments with his wife and four of a family. Ill for nine or ten months. Died March 30, 1882, from phthisis. No trace of phthisis in the family till then.

J. H., aged 18, ill for thirteen weeks, and died on March 15, 1882, from phthisis.

J. H., aged 53, wife of A. H., ill for about three months, and died on September 28, 1882. No trace of phthisis in her family, so far as could be ascertained.

G. H., aged 16, daughter of A. H. and J. H., ill for five or six weeks, and died on December 30, 1882, from phthisis.

J. H. and the mother frequently occupied the same bed, and after the death of J. H. the mother and G. H. slept together.

179.—A female recently died here from phthisis, which began soon after the death of her sister, on whom she had been in close and constant attendance for about six months.

The father and mother have died within the last two years in advanced age. No phthisis in family.

180.—I am attending an unmarried lady, living with her father and mother. She has had phthisis since January, 1882. At the middle of the year a younger sister, aged 34, came to live in the same house. Although not robust, her health was good, but in November symptoms of phthisis began to declare themselves, and both the lungs and larynx rapidly became infiltrated with tubercles, and she died January 5, 1883. The elder sister still lives.

181.—In the year 1882. Mother to child. One aunt died of phthisis.

The mother was ill for two years. The child, aged 7½, was ill two months, and died a short time before her mother. She was only ill two months. Both lungs affected. Occupied the same bedroom.

182.—One case, end of 1881. Sisters. The disease was before unknown in the family, and widespread inquiries have since failed to discover any history of it, even among the more distant relations.

183.—One case during thirty-six years of general practice, in 1866. The wife of a clergyman died eighteen months after her husband. The husband, with distinct family predisposition, came home from Bermuda invalided. Wife's family, no trace of predisposition. The wife diligently nursed the husband throughout the illness, and shortly after his death showed the first symptoms.

One daughter, aged 24, died September 5, 1881, of consumption. A younger sister, who attended closely on the one that died, has since been under treatment for allied or initiatory symptoms.

184.—(1.) In October, 1871, Mr. H., aged 48, had hæmoptysis, and again in 1873. He recovered, and is alive. His son, aged 20, died on September 22, 1872, from phthisis. The mother, who nursed him, commenced to be ill within a few months of his death, and died of phthisis on May 20, 1874, aged 50. Other children died of phthisis.

The grandparents on the father's side both died of phthisis. No history of any case on the mother's side.

(2.) Mr. C., aged 35, died of phthisis, on May 3, 1879, after two years' illness. His

mother and two or three of his brothers and sisters died from the same. He was attended entirely by his wife, who began to be ill in the autumn of 1879, and died in October, 1882, from phthisis, aged 33. Her parents are alive and well. There is no history of any tubercular disease on her side whatever.

185.—February, 1883. Patient, a boy 17 years old, in advanced phthisis. Contracted disease from his brother, with whom he had slept, and who died twelve months since. No other case in the family. Father and mother alive and healthy. Grandfather alive and healthy—says grandmother died at an advanced age. No appearance of the disease among his relatives.

186.—(1.) A man, perfectly sound, married a wife suffering from phthisis. This man died of acute phthisis some few years after marriage. No history of phthisis in the man or in his family.

(2.) A sister from another sister, through being in constant attendance on her and sleeping with her. No history of father or mother suffering from phthisis.

(3.) Mother from daughter. Mother in daily attendance on daughter. No history of phthisis in her father or mother.

187.—In 1876, in a family connection of my own, the first case, a daughter aged 21. Supposed exciting cause, a chill. The mother and brother specially paid great attention in nursing her. A few months after the beginning of her illness, the brother, aged 19, became phthisical. The two died within two or three months of one another. Shortly after this, the mother, aged 50, who had most diligently nursed her children without intermission, sickened and died of the same disease in about eighteen months.

Family history. Husband's side very good, no history of phthisis. Mother's side: her father and mother lived to above 80; no phthisis in the children.

188.—(1.) J. McI., aged 21, student, son of healthy parents, both living, no family history of phthisis, took ill with a cough while studying at Glasgow in 1870, neglected it, came home at end of session, cough got worse, found both lungs tuberculous, died of phthisis in nine months. Had one sister, 19, and one brother, 17. The former, a perfectly healthy girl, nursed her brother closely, got ill shortly after his death, and was dead in five months after him of phthisis. The brother, who slept with J. McI. during part of his illness, and wore his clothes after his death, showed symptoms of failing health before the sister died, and in about eighteen months died of phthisis.

Parents still living, 1883.

(2.) J. M., tailor, aged 34; died of chronic phthisis, in 1882. Nursed alone by sister, aged 27, one of the strongest and healthiest girls in the place. This sister complained of pain in left side about six months before her brother's death, and shortly afterwards was seized with symptoms of acute tuberculosis of left lung, of which she died in nine weeks, some three months before her brother.

I know all the family. Some of the grandparents are still living, over 90 years old. Not one of the family ever had any symptoms of phthisis.

In my boyhood pulmonary phthisis was exceedingly rare here (see Christison's report), but now it is very common. I believe in the contagiousness of phthisis as much as I do in that of enteric fever.

Both the above instances were without the slightest family predisposition.

189.—(1.) A niece, who became ill and died of pulmonary phthisis after nursing an aunt, and sleeping in the same bedroom. The aunt, an old lady, also died of phthisis.

(2.) A wife, who was quite healthy when I was called to see her husband, suffering from phthisis, but also from chronic pyæmia with abscesses. The wife became ill, and died of phthisis before the husband.

190.—I am attending a case now, March 30, 1883. The mother has developed phthisis five months after the death of one of her children, a little boy who was ill of phthisis for seven months, and died in Oct., 1882. The mother was in constant



attendance upon him, and nursed him much on her knee in a hot room. She was in perfectly good health, she says, up to that time.

191.—(1.) Some years ago the wife of an officer left Calcutta for Southampton in a sailing vessel with her husband. She stated herself to have been in perfect health when she stepped on board. The husband was in an advanced stage of consumption, and died at sea. The voyage was stormy, the hatches down, the cabin hot and close. I saw her three days after her arrival at Southampton, with both lungs stuffed with tubercles. She was an only child, with no hereditary predisposition.

(2.) Oct., 1882. A child, aged 4, in robust health, came from Berkshire into Devonshire to visit her aunt, about 30 years old, who was in an advanced stage of consumption. The child slept with her aunt, and in less than three months died of rapid pulmonary phthisis. Her brothers and sisters all healthy.

192.—July, 1882. Mother, several years a widow, aged 48, showed distinct symptoms of phthisis about three months after decease of daughter, and in six months afterwards died of it. The daughter, aged 21, had had vomicæ, with offensive sputa, and died of phthisis after two years' illness. During the whole of that period mother and daughter had slept together in a small room.

Husband died from accident. Parents of mother lived to 70 and 60 respectively. One brother died aged 36, "with all the appearance of decline."

193.—(1.) In one family six deaths, grown up brothers and sisters. Father and mother alive and hearty.

(2.) Another family, four deaths, including father. Interval between deaths variable.

(3.) A family, mother and nine children, died of phthisis. Mother's family said to be consumptive. Father alive, aged 70.

1. A son died at 21 of tubercular meningitis. First affected.

2. A son died at 17 of phthisis.

3. A daughter died at 39 of phthisis. Married. Had four children, who died young of lung complaints. Was well up to eight months of her death: when she came up to see her sister, No. 5, who was dying of phthisis. It was thought she caught cold on the return journey.

4. A son died, aged 2, of phthisis.

4. A daughter died at 33 of phthisis. Attended on No. 1 in his illness. Married. Had two children; one died of morbus coxæ, and lardaceous organs.

6. A son died at 37 of phthisis. Ill six months. Died 1881.

7. A son died at 36 of phthisis. Slept with No. 6. Well up to nine months of death. A sailor. Died 1882. Was married. His wife died of phthisis after several miscarriages. She was said to be consumptive before marriage.

8. A son died at 21 of phthisis.

9. A son died at 23 of phthisis.

The mother of the above died at 50 of phthisis, surviving Nos. 1, 2, 4. The whole family were apparently in robust health, remarkably intelligent and good-looking. Their duration of illness ranged from ten weeks in No. 4 to nine months in No. 7. I must add that the father slept with all at times.

As far as I can gather they died in the following order:—Nos. 1, 2, 4, mother, Nos. 5, 3, 8, 9, 6, 7, and were affected in the same order.

There is no doubt that the above show hereditary predisposition.

(4.) An infant died of phthisis. Sister and brother suffering from pertussis at the time. Living in same ill-ventilated room. Have both taken on phthisis. Mother suffers from scrofulous glands and corneal ulcers. Father healthy. Communicated? or common source of infection?

The subjects I refer to are poor, and live in one or two rooms.

NOTE.—It seems to me that phthisis may be communicated though the stage of incubation may be long, or the time between receiving and manifesting the virus long and variable. Hydrophobia has the same peculiarity. It does not follow that because a parent develops phthisis his offspring cannot bear witness to the communicability of phthisis. The question resolves itself into Is it hereditary? Is it communicable? or

both. If the affected parent be alive after the birth of the child there is possibility of communication.

194.—In 1862 a servant came home to her mother (a widow, with three sons and two daughters, all grown up, father dead of epithelioma), suffering from phthisis. The house, consisting of two rooms and an attic, and lying under the brow of a hill on its northern aspect, was ill-ventilated and worse lighted. By the end of 1868 the only survivor of this family, she being still alive and healthy, was a thin, delicate girl, who took little or no part in the nursing. They all died of phthisis, the mother dying last, between 50 and 60 years old.

195.—January 3, 1883. Mrs. E., who has distinct signs of phthisis at the right apex, for some weeks previously to above date slept with her stepdaughter. This stepdaughter (ill for three years) had both lungs extensively affected, and died of phthisis on January 16, 1883.

I can discover no family predisposition whatever. Mrs. E.'s first husband died of consumption seventeen years ago, and her only daughter by him, aged eighteen, died of the same complaint twelve months since. This daughter, like her mother, is said to have enjoyed good health till after sleeping for some time with the stepdaughter.

196.—A young man, of the Indian navy, came home suffering from phthisis. In a few months two of his sisters were taken with the same complaint, and died. A third sister married, and soon afterwards died of the same complaint. The young man also died. Later on the father was similarly afflicted, and died. After his death the widow became phthisical, and died also. I should think four years covered the whole outbreak—that is, from the arrival of the son from India.

The father was originally a very healthy, strong man, and all the children healthy up to about 20 or 21, or even later. I had known them all from infancy. One sister still lives, and is now between 40 and 50.

197.—April, 1881. Sisters, in affluent circumstances. The elder died of phthisis in 1879, and her younger sister, who was with her throughout the illness, died in 1881 of the same.

No family predisposition.

198.—S., a young man of 20, died twelve months since of pulmonary phthisis. During his illness he would insist upon having his half-brother, 10 years old, to sleep with him. Shortly after his death symptoms of pulmonary phthisis developed, and the lad is now in almost the last stage.

No other members of the family, consisting of eleven, have been affected, nor parents nor grandparents on either side.

199.—On this day (February 5, 1883) I attended the funerals of two sisters, aged respectively 28 and 24, the younger of whom died on the 2nd inst., and the elder on the 3rd inst. The elder sister, M. A. T., first came under my observation on September 13, 1878, with phthisis. With care and treatment the patient tided over each winter since, pulling up a little in the summer months. Twelve months ago the younger sister, M. T., who was in constant attendance, was attacked suddenly with hæmoptysis, and from this beginning of trouble phthisis progressed more rapidly than in her sister's case, and she died on the 2nd inst., one day before her sister.

The family history is good. Father over 80, mother over 70, in good health. There are three sisters living, no brothers. No trace of family predisposition.

200.—A family, named G. Husband a delicate man, with a doubtful history of phthisis in his family. Wife a strong, hearty woman, with no history of phthisis for at least three generations. They had two daughters and one son. The son died at 18 from an accident. The younger daughter died in October, 1879, aged 18, from phthisis. The father shortly afterward had a bad attack of bronchitis; he subsequently became phthisical, and died in September, 1880, aged 50, of phthisis. The mother, during her husband's illness, for the first time complained of cough and spitting of blood. She died in April, 1881, aged 53, of phthisis.

201.—E. H., a woman, aged 57, died on April 30, 1881, of phthisis. She was bedridden for some weeks previous to her death, and was carefully attended by her daughter, who slept in the same close, badly-ventilated room, and could scarcely ever be induced to leave her mother. A week before the mother died I noticed that the daughter looked very ill, and on examination she exhibited all the symptoms of acute pulmonary tuberculosis, pulse 140, temperature 103°, and loud bubbling crepitation over the lungs, but without cough or expectoration. She died on October 30. The rest of the family are healthy.

202.—From one sister to another, ages about 18 and 20. The duration in each case just twelve months. The second began immediately after the death of the first, having slept with and attended her. No other case in the family; apparently not hereditary. Parents healthy.

203.—(1.) About three years ago the wife of a consumptive husband died after about twelve months illness of phthisis. No evidence of phthisis in wife's family for generations. Herself the picture of health.

(2.) The daughter, a strong, healthy girl, shortly after death of mother, became phthisical and died in about a year after the mother. The husband survived the wife and daughter some time and died.

(3.) A wife died of phthisis. Shortly after her death the husband presented signs of the malady, and is now in an advanced stage of phthisis.

204.—A phthisical patient communicated the disease to his wife, a healthy person. Her sister nursed her, and she also became affected. A second sister nursed the last-mentioned, and she also became affected, and communicated the disease to a daughter who nursed her, and they all died. The parents and grandparents were all healthy on both sides, and lived to a long age.

## GROUP B.

## Section II.

NAME AND ADDRESS OF OBSERVER.	NO. OF RETURN.
Gason, John, F.K.Q.C.P.I., Rome . . . . .	205
Daly, G. H., M.D., Chippenham . . . . .	206
Green, J. Lardner, M.R.C.S., Salisbury . . . . .	207
Hammond, William, L.R.C.P., Nuneaton . . . . .	208
Johnson, Zachariah, F.R.C.S.I., Kilkenny . . . . .	209
Rieh, A. Creswell, M.B., Liverpool . . . . .	210
Collenette, B., M.D., Guernsey . . . . .	211
Marehbank, John, M.B., C.M., Leadhills, N.B. . . . .	212
Bindley, Philip, M.B., Bournemouth . . . . .	213
Rae, George, M.B., C.M., New Pittligo . . . . .	214
Muggeridge, Henry H., M.R.C.S., Ashford . . . . .	215
Gosling, Samuel F., M.R.C.S., Biddulph, Congleton . . . . .	216
Manley, H. C., L.R.C.P., Whitehouse, Belfast . . . . .	217
McKenzie, W. S., L.R.C.P., Larkhall, N.B. . . . .	218
Anthonisz, P. D., M.D., Galle, Ceylon . . . . .	219
Gaddy, N. D., M.D., Lovett, Indiana, U.S. . . . .	220
Gimez, Carlos, M.D., Port of Spain . . . . .	221
Vines, H. J. K., F.R.C.P., E., Littlehampton . . . . .	222
Ruddock, R. B., M.R.C.S., Clifton . . . . .	223
Foulds, Matthew, L.F.P.S., Mauchline, N.B. . . . .	224



NAME AND ADDRESS OF OBSERVER.	NO. OF RETURN.
Murray, J. Carrick, M.D., Newcastle-on-Tyne . . . . .	225
Davys, F. J., F.R.C.S.I., Cremona, Swords . . . . .	226
Small, T., L.R.C.P., Boston . . . . .	227
Moorhead, T. H., M.D., Errigle, Cootchill . . . . .	228
Farquhar, James, M.D., Harrogate . . . . .	229
Mackenzie, A. R., M.D., Fortrose, N.B. . . . .	230
Christie, James, M.D., Glasgow . . . . .	231
Caldwell, John, L.R.C.P., Shotts, N.B. . . . .	232
Mackintosh, M., M.B., Mortlake, S.W. . . . .	233
Stone, Ralph, L.R.C.S., Omagh . . . . .	234
Cornwall, John, M.R.C.S., Mearc, Glastonbury . . . . .	235
Tuke, Montague, M.R.C.S., Sutton Valence, Staplehurst . . . . .	236
Townsend, Thomas Sutton, F.R.C.S., 68, Queen's Gate, W. . . . .	237
Barr, James, M.D., Liverpool . . . . .	238
Knaggs, Samuel, M.R.C.S., Huddersfield . . . . .	239

205.—Having the medical charge of a hospital and dispensary for Italians the last three years in Rome, I have had, and still have, a great opportunity of watching pulmonary phthisis. I have had several cases under my care where the disease appeared to have been communicated from husband to wife, and *vice versa*, from brother to sister, and *vice versa*. So prevalent in Rome is the idea of its communicability, that at the hospital of St. John Lateran for women there is an upper ward reserved solely for such cases, which will not be admitted into the general wards. When a poor person dies in Rome of that disease at their own home, the family almost invariably leaves it and goes into another apartment. The greatest terror exists among the poor as to the communicability of the disease. It is met with most frequently as a sequel of bronchitis, not from hereditary disposition, and I believe it is caused by the poor inhabiting rooms which have been previously occupied by phthisical people, and which have not been cleansed.

206.—In 1878-79, four sisters. The parents are healthy. No predisposition traceable on father's side. Mother's sister said to have died from phthisis.

207.—During the thirty-six years that I have been in practice I have observed several cases of phthisis which appeared to have been communicated from a sick person to the nurse, generally a mother or sister.

208.—I have seen such cases, but can give now no details.

209.—My uncle, an eminent Dublin practitioner, used to quote the case of the mother of a large grown-up family, who, in his belief, distinctly contracted phthisis from her eldest daughter, who died of that disease. So far as I know, no other member of the family developed it.

210.—Dec. 1882. A case under my care at the present time of phthisis in a man aged 21, whose sister died of same disease a few weeks ago. No family predisposition. House small, with close, ill-ventilated kitchen in which the brother and sister used to sit.

211.—I have seen several cases between husband and wife and between sisters. There was no family predisposition, in these cases, so far as could be ascertained. The last case of this class, two sisters, happened about three years ago, the father, mother, grandfathers, and grandmothers, all being alive.

212.—Two cases. Two years ago. Sister and mother of patient. No reliable information obtained as to family predisposition. They lived in one apartment. The

sister first, and next the mother, became affected within a few weeks after the death of the first patient. Two or three brothers and the father, who spent most of their time in the open air, escaped.

213.—Dec. 1882, a lady, aged 28, with laryngeal phthisis. Father and two brothers died of phthisis, aged 59, 18, and 22. Nursed her youngest brother who died of phthisis, sat beside him on sofa, read a great deal to him. Illness began a week before his death.

214.—The niece, *æ.t.* 15, of a man 35 years of age, who died of phthisis in 1882, had been in almost constant communication with him during his illness, shewed symptoms of phthisis in February, 1882. No family predisposition ascertainable.

215.—(1.) In 1872, wife from husband.

(2.) In 1878, married woman, aged 32, died of phthisis on October 5. Her unmarried sister attended upon her and nightly slept with her. Attended also to her family. Was perfectly well before. Father and mother both living, none others of the family were consumptive. This young woman married, was twelve months ill, and died *æ.t.* 21, on November 29, 1879.

216.—Have observed many cases of communication, but always in same family, and always hereditary taint.

217.—(1.) The wife died of phthisis, and the husband who was in delicate health at the time of his wife's death, died of well-marked phthisis about a year and a half afterward. Was in constant attendance on his wife.

(2.) Two brothers died of phthisis. Sister alive, in delicate health. Tubercular deposit at apex of left lung. Mother died of chronic bronchitis and emphysema. The family all lived together.

(3.) Large family, six or seven brothers and sisters, all died of pulmonary phthisis. In the last the disease was very acute. The family all lived together.

218.—1878. Three brothers and sisters died within sixteen weeks of phthisis, a few months afterwards another sister died, surviving sister phthisical. Mother (family predisposition) died of phthisis in 1882.

219.—(1.) A clerk in the Colonial Office, at Colombo, Ceylon, a widower, with three children, married a second time a lady of consumptive family, who died shortly after marriage of consumption. Her husband, who was quite well at her death, and who attended, nursed, and occupied the same room with her until shortly before her death, became consumptive a few months later, and died, as far as I can remember, within two years of the wife's death. Had no children by the second marriage. Those of the first wife are alive, well, married, and have families.

(2.) The wife of a young man who died of consumption, had nursed him, and took ill after his death, and died from it, and his sister, who nursed her sister-in-law, also was affected and died.

220.—(1.) Jan. 25, 1883. A young lady now under treatment, nursed a lady having phthisis two years ago, and has been declining ever since. Parents both living and aged.

(2.) A woman, aged 25, recently died of phthisis, whose husband died of same one year ago. Before his sickness she was healthy, no consumption in the family, but a brother of deceased is now suffering from it, said to have caught it from another person where he lived a few months.

(3.) A woman of a healthy family, also fell a victim to it a year after her husband died of consumption.

221.—1875. Sister from brother, whom she nursed; began to be ill a month before his death, two attacks of hæmoptysis, died in about seven months. 20 years of age. Father and aunt died of phthisis.

222.—I have seen such cases, but neglected to keep notes.

223.—I have no notes to go by, but I have long considered that pulmonary phthisis was highly contagious.

224.—I have seen more than six or seven cases.

225.—I have seen cases between husband and wife, also of brothers sleeping together. I have a marked case now in my mind where there is no suspicion of heredity. The elder brother died August 11, 1881, of phthisis. His brother, and bedfellow until I separated them, is now wintering in the Mediterranean, with tubercle in both apices.

226.—Have seen symptoms of phthisis develop in husband (surviving wife) and daughter (surviving mother) without predisposition, but following on close intimacy.

227.—I have thought so with sisters; but kept no record of the cases.

228.—(1.) In December, 1868, I came home from Dublin in good health. My father was suffering from phthisis. I nursed him for three months, being constantly with him in a warm room. At the end of six weeks I got a cough, followed by hæmoptysis, and a cavity in the right apex, which was cured by a sea voyage. The disease in my father's case was the sequel of pneumonia, and I had no hereditary tendency on either side.

(2.) In December, 1878, Dr. M., a brother practitioner, got pleuritis, with effusion. He recovered, but his cough continued. In February, 1881, his wife died of chronic phthisis, and in the following April he died of tuberculosis. There was no hereditary tendency in his case.

\*229.—Mr. G., rector of F——, had a son (captain in army) who nursed a wife through acute phthisis, contracted it quickly after her death, and died of it after a very short illness. No hereditary taint at all in the G. family.

230.—A. G., a mason, died, aged 24 years, on May 17, 1881. His mother, M. G., who had constantly attended him during his illness, immediately afterwards began to show symptoms of phthisis, which ran the usual course, and she died, aged 68, on December 26, 1882. Both herself and her family had previously been healthy.

231.—While acting as dispensary surgeon and physician in the Western Infirmary, Glasgow, numerous cases came under my observation.

232.—Three cases, in cousins. No family history of phthisis. Slept in the same bed for three weeks.

The first in November, 1881; the second in February, 1882: the third in December, 1882. The first two are dead; the third still under observation.

233.—A man died of phthisis in October, 1881. His brother-in-law, 26, was in the habit of inflating an air bed on which the patient lay by blowing into it. Shortly after the decease of his brother-in-law, my patient began to present the symptoms and physical signs of phthisis August, 1882. He has since, however, to a great extent recovered. No history whatever of phthisis in his family.

234.—Three cases within the last twelve months. The relationship was in all cases brother and sister, with a family predisposition to the disease.

235.—Three sisters; one in 1841, another in 1842, and the third in the following year. The predisposition was on the mother's side.

236.—A girl, aged 30, nursed a brother for two months, who died last November of phthisis. The girl was, up to the time of her brother's illness, fairly strong. She nursed her brother very carefully. Father died of phthisis at 45; mother alive and

\* Should be in Group A.



well, aged 74. The present stato of tho girl is as follows :—Profuse night sweats, catameuia entirely ceased, pulse 120, temperature 99·2°, left lung rapidly breaking down. I have not the slightest doubt that, though the girl was somewhat predisposed to phthisis, the nursing her brother last August and September set up active disease.

237.—I have seen many cases, I should say ten, if not twelve, over a period of twelve years.

238.—One case at present under observation. Two sisters, one of whom died last week. No family predisposition.

239.—I have seen several cases. I have not made special notes of the cases, but I have no doubt as to the facts.

### GROUP C.

*Returns containing cases observed between persons unrelated as well as between members of the same family.*

NAME AND ADDRESS OF OBSERVER.	NO. OF RETURN.
Straton, C. R., L.R.C.P., Wilton . . . . .	240
Thomas, J. Raglan, M.R.C.S., Clanelly . . . . .	241
McClelland, R. B., M.D., Banbridge . . . . .	242
Jackson, E. S., M.B., C.M., Carnforth . . . . .	243
Whittaker, W. M., M.B., Valentia . . . . .	244
Holbertou, Henry N., L.R.C.P., Hampton . . . . .	245
Vesey, T. Agmondisham Blathwayt, M.K.Q.C.P., Knapton . . . . .	246
Maguire, T. S., L.K.Q.C.P., Stony Stratford . . . . .	247
Lowther, Richard, M.D., Cartmel, Grange-over-Sands . . . . .	248
Simpson, James, L.R.C.P., Edinburgh . . . . .	249
Juler, Henry Cundell, M.D., Cincinnati, Ohio, U.S. . . . .	250
Dalton, Charles G., M.R.C.S., Lincoln . . . . .	251
Mayer, A., M.D., Antwerp . . . . .	252

240.—(1.) William H. returned from London in December, 1881, and died of phthisis in February, 1882. His sister, who nursed him, though previously in good health, took the disease, and died in October. Her chief companion, a girl in excellent health, who devoted herself to her in her illness, has phthisis now, but is still alive. Her first hæmoptysis was just before her friend's death and while waiting on her. Parents of all alive and well.

(2.) E. S., aged 28, died of phthisis September, 1881. She was visited constantly by her cousin, A. F., who developed the disease, and died in February following. A. F.'s brother and sister have since developed the disease, but are still alive, while another sister of A. F., who likewise took it, died in February, 1882, leaving an infant, which likewise died of marasmus eight weeks later. E. S. also left an illegitimate child, which died also of marasmus.

Parents of E. S. and A. F. alive and well.

241.—In 1876 Mr. —, a solicitor, died of phthisis, contracted from his wife. His family history was exempt from taint of phthisis, both parents being still alive at a great age.

A servant, who had nursed both devotedly, died soon afterwards: but in her case there may have been a constitutional predisposition.

242.—(1.) J. K., aged 22, died about March 25, 1859. All his relations were healthy. His father is still alive. He always had good health until he went to the seaside in 1858. There he slept with P. C., aged 21, who was at that time ill with phthisis, and died February 3, 1859.

(2.) P. G., aged 46, died July 20, 1878. Health was always good until she commenced to sleep with and attend her daughter, who died of phthisis on March 15, 1878, aged 23. P. G. was the youngest of a family of eleven, who were all healthy, and of which a good many are still living. Father and mother both lived to above 80.

243.—(1.) H. D., 38, infection from child. His family very healthy, and no hereditary history. Himself a very healthy, strong man.

Nursed child very closely. Child died. He is still living, although cavities in both apices. Now in Davos Platz.

(2.) Reports also another case between two friends.

244.—About five years ago the wife of a butcher living here died of phthisis. A few months after her death the disease became well-developed in her husband, a man of very powerful physique and hitherto free from any ailment. He died after two years. I heard his brother died some years before in London of what was said to be a galloping consumption, but I don't know whether it was so or not. A short time after the man exhibited signs of the disease, he employed a young man of 18 as an apprentice. He after a short time complained of cough and hæmoptysis, and in about a year and a half died of well-marked phthisis, his father, mother, brothers, and sisters, all being quite healthy. I should add that the apprentice slept with the butcher, and was healthy till he went into his employment.

245.—(1.) 1880. A young man, previously healthy, engaged to be married to a girl afflicted with phthisis. The engagement lasted about three years, and the parties were constantly together, until the girl died. About three months afterwards the man presented symptoms of acute phthisis, and speedily died.

Two sisters of the girl died of phthisis. There was no family history of it in the case of the man.

(2.) June, 1882. Husband and wife. Wife markedly phthisical, with cavities in both lungs. Been married some years. Husband developed symptoms in June last.

No history on husband's side of family.

(3.) May to October, 1882. Husband and wife. Wife phthisical. Husband syphilitic, but no family history of phthisis. Married seven years. Husband then developed symptoms of phthisis (? syphilis of lung), and died in six months.

246.—(1.) August, 1867. The disease was communicated to a young girl of 20, who was attending to a case of phthisis in male subject. No relation. A brother of the girl's died of hydrocephalus in 1869. Parents both alive. No phthisical history in the family. Rapidly fatal.

(2.) April, 1876. Communicated by an elder sister to a younger, and strange to relate both cases recovered, though there were well-marked physical signs. Family history good. Mother died, I am informed, from heart disease. Father died from acute pneumonia and mitral disease.

247.—Five well-marked cases of communicated phthisis within the last ten years. Case 1, husband and wife; 2, ditto; 3, sisters; 4, patient and nurse; 5, ditto.

In two of the cases at least there was certainly a consumptive tendency, there was also close confinement to badly ventilated and small rooms.

248.—(1.) Miss N., aged about 38, married a clergyman, the year afterwards he became phthisical, his case being a very chronic one. The wife's health, previously good, gave way eight or nine months after her husband's illness commenced, in April, 1873, and she died of phthisis in August, 1875.

No hereditary tubercular tendency. The husband still lives (January, 1883), though in advanced phthisis.

(2.) Mrs. M., age about 26, a farmer's daughter, very robust and healthy, married a man who became consumptive after birth of first child. His wife nursed him till his death in 1878. Immediately afterwards she became phthisical and died in 1882.

Some scrofula in her family, but no deaths from phthisis before her own.

(3.) A maidservant, aged 16, whose health had been previously good, nursed her mistress, who was phthisical, till her death, sleeping in the same room with her. A few weeks after that event I found the maid distinctly tubercular. The disease rapidly advanced, but I lost sight of her, as she left the district for her own home.

NOTE.—In each of the above cases, the nurse slept not only in the same room, but in the same bed with the patient.

249.—(1.) A woman with acquired phthisis appeared to have communicated it to two sisters, the first, married, with whom she came to reside when she first was taken ill. Afterward to another with whom she went to live 400 miles away. Date of observation, 1882. No history of phthisis in family.

(2.) A strong robust young man married a widow whose first husband died of phthisis. He became phthisical within twelvemonths after marriage. No history of phthisis in his family. The widow herself has chronic phthisis. Date of observation, 1882.

(3.) A young man of 18, with good family history, died of phthisis, which appeared to be contracted by close contact with a fellow-workman who had phthisis. Date, 1881.

250.—In 1854 I treated a Mrs. G., in Isleham, Cambs., with phthisis. Her relatives were healthy country people. She nursed a lady who died of phthisis. After the death of Mrs. G., her sister, who had nursed her, fell a victim to the disease.

251.—(1.) Husband, with no family history of disease, became phthisical during illness (fatal) of wife from this cause, and eventually died *et.* 32.

(2.) The second wife of a labourer, no family history of disease, died of phthisis after nursing a daughter of the first wife (who had died of phthisis some years previously), during last fatal illness from consumption.

(3.) Observed in 1881. A healthy servant girl, aged 19, living at a country rectory, and who slept with a fellow servant girl for several months, the latter leaving her situation on becoming phthisical, died in the summer of 1881 of phthisis.

No other member of her family affected before or since.

252.—(1.) In 1840 died a married lady, issued of a tuberculous family. Her husband, with an excellent family history, became soon after his wife's death consumptive, but married again a young lady, in whose family phthisis had never occurred. The gentleman died consumptive in 1842, and his second wife took the same disease and died in 1843.

(2.) A lady, aged 28, who had lost her parents and six brothers and sisters by consumption, was governess in a family with an excellent history. One daughter was much attached to the governess, slept in the same room, and from time to time in the same bed with her, and never left her till her death in 1849. The young lady, aged 20, became tuberculous, and died in 1851. Her parents, her three brothers and her sister, are alive yet, and quite healthy.

And several analogous cases.



## GROUP D.

*Returns containing cases observed between persons unrelated only.*

NAME AND ADDRESS OF OBSERVER.	NO. OF RETURN.
Carter, D'Arcy B., L.R.C.P., Wakefield . . . . .	253
Barrow, R. W., L.R.C.P., Liverpool . . . . .	254
Sprigge, Charles, M.D., Great Barford . . . . .	255
Blake, G. F., M.R.C.S., Bishopstone, Moseley, Birmingham . . . . .	256
MaeDowell, Charles, M.D., F.R.C.S.I., Carlow . . . . .	257
Adkins, Edward J., M.R.C.S., Hastings . . . . .	258
Browning, Benjamin, M.D., Rotherhithe, S.E. . . . .	259
Fitzmaurice, N. F. H., L.R.C.P., Dunning, N.B. . . . .	260

253.—Six months ago a woman, aged 35, was found to be suffering from phthisis. She had been quite well up to the time that she went into service at a house where there were two phthisical patients, and it was part of her duty to empty and clean the spittoons of the patients twice a day. No relationship.

I could not make out any family history of the disease.

254.—Jan., 1882. A girl, aged 19, acted as maid and companion to a lady who was the subject of chronic phthisis. The girl was in the habit of sleeping with her mistress every night for a considerable period. Previous to her engagement she was in robust health. When I was called in I found her suffering from acute phthisis, which ran a quick course, and was rapidly fatal. No relationship and no family predisposition.

255.—Miss R., aged 48, a dressmaker, living in rather a lonely cottage at C., Bedfordshire, had three apprentices, young girls of from 17 to 19 years of age, not related, from three adjoining villages, who took it in turn to remain in the house and sleep with her, each one for a week at a time. During their apprenticeship Miss R. was taken with phthisis, of which she died. In less than two years afterwards all three apprentices died of phthisis, although in the family history of each no trace of phthisis existed, and the parents, brothers, and sisters of two of them are alive and well at the present time.

256.—May 14, 1882. No relationship. No family history of the disease in parents, grandparents, &c.

A perfectly healthy child until taken care of by a nurse suffering from pulmonary and laryngeal phthisis, who had the absolute care of the child night and day. The room, which was used for day and night nursery, was warm and close. The disease ran a very rapid course in the child, with rather high temperature, and ended abruptly in a copious hæmoptysis.

257.—In one particular ward in our local asylum, with 350 patients, phthisis has its habitat to the exclusion almost of the other divisions. Certainly we have had one or two sporadic cases in other parts of the institution, but in no instance did the malady seem to hold on, so to speak, or to select one part of the building, save in this. The keeper of the ward in question had been suffering from phthisis for years past up to the 2nd inst., when he died. Along with him we have had several of the patients attacked, keeping up a supply of the disease, though other portions of the house were free from it.

258.—1880. A young woman, 18 years old, somewhat delicate, attended occasionally as nurse a case of phthisis in the last stage, no relation, and after he died she complained of having inhaled his breath and odour of the room, the feeling of which she could not get rid of. After a short time phthisis became developed, and she succumbed to it after an illness of nine months.

There is no history of phthisis in the family, father and mother being still alive and quite healthy, except one of her brothers, late a sailor in the Royal Navy, who has recently died from phthisis, contracted abroad after an attack of pneumonia, brought on by sleeping out of doors in the wet while in a drunken fit.

259.—I saw several cases, occurring in a line-of-battle ship, the *St. Jean d'Acre*, belonging to the Mediterranean fleet in 1860–61. I visited her from time to time, and can corroborate the account as detailed in the statistical report of the health of the Navy for those years.

260.—A female aged 24. Mother and father both alive and well, but all the maternal aunts died of phthisis. Before her confinement she had slept constantly with a person, no relation, who had phthisis. She was before this free from all symptoms of disease.

She died Feb. 1883.

261. Tables and Letter of Dr. Leith Napier.

#### DR. LEITH NAPIER'S LETTER.

SIR,—Influenced by a desire to contribute to the present inquiry regarding the nature of phthisis, I send two tables of the life history of a family of which I have accurate and intimate knowledge.

These tables show (1) the hereditary nature, or the predisposition by heredity; (2) the malinfluence of intermarriage of individuals with phthisical family histories [no one of the persons on the table was known to have developed symptoms of phthisis before marriage, so far as I can ascertain]; (3) the infectious or rather contagious nature of the disease, as shown by A (i.), Mr. and Mrs. K.; A (ii.), Mr. and Mrs. T.; A (iii.), Miss N.; and A (i.) 2, Miss A. K., &c.; (4) the tendency of tubercle to show itself as meningitis in the offspring of phthisical ancestors, as in A (ii.) 3, and (iv.) 4, 9, 11: (5) the beneficial effect of a healthy husband or wife as a preventive against phthisical offspring [that the husband should be healthy is possibly the more important, but this is not as yet determined definitely]; (6) how the disease may pass a generation, or individuals, of a family, other members of which are affected.

Of a certainty no one of these points is novel, or even disputed by the great majority of observers. With the exception of the influence of the male or female parent, there can now be no variance. I think a phthisical mother affects the offspring more markedly as infants, a phthisical father at a later stage, but no less surely.

As an actual record of reliable personal information and observation by a family practitioner, I hope the tables may be thought worthy of acceptance.

I trust others may proceed on the same lines, and report to the Committee like statistics. I doubt not hundreds or thousands of such tables might be written, and if skilfully summarized would prove of great value in settling our theoretical ideas on a firm practical basis by recent observations.

Yours faithfully,

A. D. LEITH NAPIER, M.D., &c.

DUNBAR, N.B.

TABLE A.—FAMILY OF MR. AND MRS. N.

I. Mr. N. died suddenly of heart disease, aged 48. No history of phthisis in his family.

II. Mrs. N. died, aged 57, of cancer. Never suffered from any chest complaint. Her brothers and sisters all healthy. But there had been a history of consumption in the family of her mother, Mrs. O.

Mr. and Mrs. N. had issue:—

(i.) Mrs. K., who married at 21, and died of phthisis at 46, four years after her husband. Mr. K. was of a consumptive family. He had fever and after it phthisis. His wife nursed him. She was then suffering from her chest. She slept with Mr. K., and possibly infected him. Mr. K. was ill a year and a half, and died of phthisis at 40.

They had issue:—

1. Mrs. B. Married at 22. Had six children. Died at 36 of consumption. Husband rather delicate.

Mr. and Mrs. B. had six children. Five are living, from 14 downwards. Delicate. One died of pulmonary disease.

2. Miss A. K., who slept with her mother, Mrs. K. constantly, and nursed her during her last illness. Died of acute tuberculosis at 19. Three months ill.

3. John K., died of phthisis, after two years' illness, at 21.

4. Mrs. A. C., married at 22 and delicate, now 28.

Mr. and Mrs. A. C. had two children:—

One died; one living, about 2.

5. Died in infancy.

6 and 7. Born after mother's first illness. Both delicate. 6. Miss I. K. 7. James, died, aged 21, of pneumonia (?), after short period of ill-health.

(ii.) Mrs. T. married at 17; died of phthisis at 26; nursed and slept with her husband when he was ill of consumption, and survived him eight months. Mr. T. had consumption in his family, and died of it after two years, aged 40.

They had issue:—

1. William, 36; 2. Daniel, aged 34. Both have had chest weakness.

3. Mrs. C. married at 22 to a husband whose brother died of phthisis. She died of phthisis at 27.

Mr. and Mrs. C. had three children:—

A baby who died at six months, a boy who died at 6, and another boy who died at 5; all of meningitis.

4. Mrs. G., married at 24 to a delicate husband. Mrs. G. has never had any chest complaint, is healthy, now 38. Mr. G. married at 26, died at 30 of phthisis after a year and a half of illness. A half sister and a half brother, one on the father's, one on the mother's side, died of consumption.

Mr. and Mrs. G. had four children, from 6 to 2. All healthy.

5. Robert, married at 24, wife healthy, but sister-in-law weak chest, brother-in-law spine disease. R. has had pleurisy more than once.

Mr. and Mrs. R. T. have had three children, aged from 7 downwards. One died of croup.

(iii.) Miss N., unmarried. Nursed her sister, Mrs. T., during her illness. Contracted phthisis and died of it about a year afterwards, aged 28.

(iv.) Mrs. R., married at 19 to a healthy husband, now 62, whose family was free from phthisis. Died at 47 from uterine pyæmia at the climacteric.

They had issue:—

Two children (1 and 2), who died in infancy. 3. A child who died of croup at 4½.

4. A baby who died of meningitis at 6 months. 5, 6, 7, and 8. Four children who are all healthy, aged from 22 to 28. Of these 5, Mrs. L., married at 23. Her husband's family are very healthy. Mr. and Mrs. L. have four children, aged from 5½ to 3 months. All healthy.

9. A child who died of meningitis at 6.

10. Aged 16, healthy, but has enlarged cervical gland.

11. Died of meningitis at 5.

(v.) David, healthy, killed accidentally, aged 26.



TABLE B.

*(Giving the same facts in columns.)*

Mrs. O.'s family suffered from phthisis. Her daughter, Mrs. N., and her other children were free of the disease.

By Mrs. N. she had 5 grand children, of whom . 3 died of phthisis, 2 otherwise.

Of the 3 husbands of Mrs. N.'s daughters . 2 " "

By Mrs. K. (Mrs. N.'s d.) she had 7 great grand children, of whom . 3 " " 1 "

Three other of the grandsons-in-law are weak-chested.

2 great grand children-in-law.

By Mrs. T. (Mrs. N.'s d.) she had 5 great grand children, of whom . 1 " "

Three others weak-chested. One healthy, but husband died of phthisis.

3 great grand children-in-law, of whom . 1 " "

By Mrs. R. (Mrs. N.'s d.) she had 11 great grand children, of whom . 3 " " 3 "

Five are healthy. One girl married, giving 1 great grandson-in-law.

By Mrs. B. (Mrs. K.'s d.) she had 6 great great grand children, of whom . 1 " "

By Mrs. A. C. (Mrs. K.'s d.) she had 2 great great grand children, of whom . 1 " "

By Mrs. C. (Mrs. T.'s d.) she had 3 great great grand children, of whom . 3 " "

By Mrs. G. (Mrs. T.'s d.) she had 4 great great grand children.

By Mr. R. T. (Mrs. T.'s son) she had 4 great great grand children, of whom . 1 "

By Mrs. L. (Mrs. R.'s d.) she had 4 great great grand children.

Total of Mrs. O.'s descendants, including their partners by marriage, 60, of whom 18 died of phthisis, 7 otherwise.

## CLASS II.

## OBSERVERS WHO ARE DOUBTFUL.

NAME AND ADDRESS OF OBSERVER.	NO. OF RETURN.
M'Lachlan, W. A., M.D., Dumbarton . . . . .	262
Fletcher, Bell, M.D., Leamington . . . . .	263
Humphrey, Lawrence, M.B., Cambridge . . . . .	265
Ransome, Arthur, M.D., Bowdon . . . . .	266
Grubb, J. Strangman, L.R.C.P., Waterbeach . . . . .	268
Armistead, William, M.B., Shelford . . . . .	270
Stewart, William, M.B.C.M., Kirkwall, N.B. . . . .	278
Brown, J., L.R.C.P., Bacup . . . . .	280
Gibson, John H., M.D., Hull . . . . .	283
McCalman, Dove, M.D., Ballachnlish, N.B. . . . .	289

And others omitted for want of space.\*

\* Exigencies of space alone have necessitated the selection of *representative* returns in this and the next class. It must be understood that those omitted are of equal importance and of similar character to those published.

262.—To this question I am inclined to answer, No ; and yet I am doubtful, from a consideration of the following cases, which came under my care in 1879, that this No should not be Yes.

*Case.*—J. M. C., a middle-aged man, a stone mason by occupation, had been ill for some time with bronchial catarrh, to which the symptoms of pulmonary phthisis became added, and were accompanied by increased temperature and sweating, to which he quietly succumbed. His wife, a comparatively healthy woman of about his own age, nursed him and slept with him during his illness, and she eventually manifested symptoms of phthisis, and died about two months and a half after her husband. A son, a boy of 14, in constant attendance on his mother, sickened after her, and in about three months died. Their daughter, a girl of 16, showed signs of being influenced by the same disease, but a timely removal to more healthy surroundings seemed to protect her. Her sister, a robust young woman, a school teacher in some part of England, came to stay for a short time toward the death of her brother, and had at her coming no signs of chest complaints. Yet she felt not quite so well during her residence here. She and her sister are at present quite well, and do not show any symptoms of phthisis. The present tenants of the house where these deaths occurred were very healthy before they went to it, but since then I have frequently seen members of the family ill with bronchitis. I write these notes purely from memory.

[NOTE.] It is a house of two apartments in Dnmbarton. I have reason to think that all of them slept in one room, thus rendering the house overcrowded. It is by no means a damp house, and not isolated. It is, however, in close proximity to the river. The drainage of it is not good, the drains communicating directly, without intervening cesspools, with the common sewers. There are no trees in its immediate vicinity.

263.—Upwards of fifty years' experience enables me to say that I have witnessed many cases of pulmonary phthisis which have *appeared* to have for the exciting cause communication with persons suffering from the same disease, in whom there has been an hereditary predisposition. As, for instance, in families, the most diligent nurse among the sisters has been the next to succumb. But I do not remember any case so caused where there has been no hereditary disposition.

265.—I have to give an account of the following case without my notes. The main particulars are, I believe, fairly accurate.

*Case.*—E. L., aged 24, nurse at the Chest Hospital, Victoria Park. Born in India. Her father, a soldier and a drunkard, died when she was young, disease unknown. Her mother alive and intemperate. E. L. left her home at an early age to escape from the family, and had seen nothing of them since, and heard nothing with regard to consumptive taint. E. L. nursed cases of heart disease, bronchitis, and consumption of all kinds. Complained of feeling ill in Jan., 1881, but had been out of health for last two or three months. This was about the end of her second year at the hospital, and she had the appearance of being strong and healthy. Latterly she had lost flesh. The physical signs were slight but suspicions of incipient disease at the left apex. A week later (end of Jan.) she was taken suddenly ill with shivers and fever. The physical signs were those of acute pleurisy all over the left lung. This was followed by rapid consolidation, softening, and excavation of the upper lobe of the left lung within two months. The disease became then to a great extent quiescent, and she gained flesh and improved. Went to a convalescent home for the spring and summer, where she first had a little diarrhoea. Returned to the hospital in the autumn. Got gradually worse : ulceration of the bowels, and progressive consolidation and softening of both lungs, and she died in Jan. or Feb., 1882, about a year after physical signs were noted. She had been a teetotaler all her life, but having no home to go to, used frequently, when she had leave from the hospital, to return wet through to the skin, and often got cold. There was no post mortem examination. There were no particular cases of phthisis under her charge noticed, but the greater number of the cases are phthisical.

266.—In the course of twenty-five years' practice I only remember one case in which phthisis seemed to have been transmitted from a husband to a wife.

I doubt whether the bacillus can germinate in a perfectly healthy individual, without the concurrence of a foul atmosphere, and a continuously high temperature.

The following case is given for what it is worth. I do not myself think it conclusive.

Mrs. B., 35, living in Manchester, an out-patient (No. 7084) at the Hospital for Consumption there, washerwoman, states that no member of her family, to ncles and aunts, has been consumptive. Was married fourteen years. Two years ago her husband died of phthisis. Five years ago she herself had bronchitis, lasting three months. Since then healthy until eighteen months ago, when she had a winter cough. She did not lose flesh until seven months ago, when her present ailment commenced. She has now softening tubercle on one side, and incipient consolidation on the other. Bacilli present in sputum.

263.—In a country practice it is almost impossible to decide between phthisis communicated and hereditary, as patients are generally nursed by members of the same family.

270.—Not having been in practice for more than ten years I can give you no recent cases. But when in Manchester, in 1870–72, I remember I had cases in which phthisis appeared to be communicated from husband to wife, or *vice versa*. Later observations of the disease, as Medical Officer of Health, have led me to the conclusion that in some of the cases in which phthisis appeared to be communicated from a delicate husband to a previously healthy wife, or *vice versa*, the disease may have been due to the same exciting cause acting quickly on the predisposed and more slowly on the previously healthy, such as sleeping in the same overcrowded bedroom, living on the same damp clay soil, or breathing cotton dust in the factories.

273.—The following is given as a suggestion with regard to phthisis and family history.

J. B. and E. B., brother and sister, both died of pulmonary phthisis. The eldest of the family, a sister, is married, and has large healthy family. Parents, grandparents, uncles, and aunts, all healthy, with no history of phthisis. J. B. married A. E. Her family history also free from pulmonary affections. A. E. pre-deceased her husband, J. B., from phthisis; he, J. B., succumbing a year and some months later to the same disease. E. B. was married to J. P. She died of phthisis. J. P. married a second time, and succumbed to pulmonary phthisis shortly after second marriage. J. P.'s family history good, and free from phthisis. J. P.'s widow has married again, and at present seems in good health. Her family history is free from phthisis. I look with some interest to the conclusion of this more than ordinary sequence of phthisical affections occurring in healthy families.

I know of many cases which negative the contagiousness of phthisis.

The above cases have ranged from the date of death of A. E. (1870) to that of J. P. (1880).

280.—No case during five years' practice, except the following cases, which occurred a few weeks ago, which may have been due to contagion or to the unsanitary surroundings and want of proper food.

Within two months two sisters and a brother died of tuberculosis. September 18, 1882, Bridget S., 2½ years, died of tubercular phthisis. November 6, 1882, Rebekah S., 17 years, died of tubercular phthisis. November 6, 1882, Patrick S., 19 years, died of tubercular meningitis. Patrick S. was well up to July, 1881. Under medical treatment until July, 1882, for ascites. Was then admitted into Manchester Infirmary. Was there for about two months. Tapped twice. Sent back no better. Was under my care, and the ascites had nearly disappeared by middle of October, when slight cough set in. No physical signs of lung deposit. Pulse was quick and weak. About five days before death an intolerable headache came on, with marked signs of tubercular meningitis, which proved fatal on November 6, 1882. The cause of the ascites was never clearly made out.

Rebekah S. was taken ill in May, 1882, with a cough. Soon after, physical signs of tubercular deposit in both lungs. Case ended fatally on November 6, 1882.

Bridget S., *et. 2½*, was taken ill with croup on August 2, 1882. She recovered from this in a fortnight, when lung mischief developed. She died on September 18, 1882, of tubercular phthisis.



Father and mother, brothers and sisters, all well. None have died of phthisis. The history of the whole case is difficult to ascertain. Know nothing of the grandparents.

This is the only case in which I have ever observed anything like evidence of probable contagiousness of pulmonary phthisis. It is remarkable that three cases should occur and prove fatal so near to one another. The elder sister and brother were well and at work in the mill until the time of illness. Were considered strong and healthy.

283.—I consider that the period of incubation may be so long, and the time elapsing up to development of phthisis so indefinite according to favourable or unfavourable surroundings, that it is possible the disease may be communicated and not observed.

289.—The above question I can answer neither in the affirmative nor negative. I unfortunately see a great many cases of this disease, and must confess that my former belief in its non-contagiousness under certain circumstances is considerably shaken. That phthisis is contagious in the case of an individual hereditarily predisposed to it, when that individual lives closely for a considerable time in a warm confined atmosphere with an individual suffering from the disease, I believe, as I have seen cases which to my mind point strongly in that direction. Still there are many sources of fallacy. But I met with no case where a perfectly healthy individual, or individual inheriting no predisposition to phthisis, contracted phthisis from another suffering from the disease, though I have had a number of cases under my care where such healthy individuals attended closely for months on cases of phthisis in very small, ill-ventilated houses.

---

Answers coming in this class have been received also from:—

John M. H. Martin, M.D., Blackburn; John W. Workman, M.R.C.S., Reading; S. Rees Philipps, M.D., Exeter; Edward Haughton, M.D., Upper Norwood, S.E.; H. Boyle Rannalls, M.R.C.S., Saltash; Algernon A. Cohen, M.B., Burwash; Kenneth W. Millican, L.R.C.P., Kineton; Thomas Lettis, M.R.C.S., Great Yarmouth; A. Mullan, M.D., Ballymena; A. M. Sculthorpe, L.R.C.P., Tamworth; William W. Ireland, M.D., Prestonpans, N.B.; Alexander John Macarthur, M.D., Anstruther, N.B.; William Rayner, M.R.C.S., Dorset Square, N.W.; Francis J. Allan, M.B., Dock Street, E.; John Martin, L.R.C.S., Surg. A.M.D., Cork; A. H. Newth, M.D., Hayward's Heath; Byron Bramwell, M.D., Edinburgh; F. Charleswood Turner, M.D., Finsbury Square, E.C.; J. Lindsay, M.D., Lesmahagow, N.B.; Ed. H. Dickinson, M.D., Liverpool; George Newstead, M.R.C.S., Ecclehill; J. R. Ross, M.D., Ballykelly; Appleby Stephenson, M.D., Nottingham; Alfred Eddowes, M.D., Market Drayton; Charles E. Addison, L.K.Q.C.P., Colechester; R. Wilson, Moore, L.R.C.P., Wednesbury; William Francis Hazel, M.R.C.S., Oakley Square, N.W.; John Wilton Sheridan, L.R.C.P., Stowmarket; J. Birkbeck Nevins, M.D., Liverpool.

## CLASS III.

## NEGATIVE OBSERVERS.

NAME AND ADDRESS.	NO. OF RETURNS.
Harday, George, M.R.C.S., West Haddon . . . . .	302
Daniel, James, L.K.Q.C.P., Cheadlo . . . . .	303
Black, W. T., F.R.C.S., Surg.-Maj. A.M.D., Edinburgh . . . . .	304
Haworth, James, M.R.C.S., Filey . . . . .	305
Bennett, A. Hughes, M.D., Old Cavendish Street, W. . . . .	306
Hall, William, L.R.C.P., Lancaster . . . . .	307
Tacey, William G., L.R.C.P., Bradford . . . . .	309
Church, W. S., M.D., Harley Street, W. . . . .	311
Row, F. Everard, L.R.C.P., Devonport . . . . .	315
Mahomed, F. A., M.D., St. Thomas Street, S.E. . . . .	316
Tatham, J., M.D., George Street, W. . . . .	317
Webb, Fredk. E., M.R.C.S., Maida Vale, W. . . . .	320
Latham, W., L.K.Q.C.P., Ashton in Makerfield . . . . .	329
Gabb, John, M.R.C.S., Bewdley . . . . .	339
Fraser, G. Ross, L.R.C.P., Wark on Tyne . . . . .	340
Garstang, T. W. H., M.R.C.S., Dobercross . . . . .	350
Allbutt, T. Clifford, M.D., F.R.S., Leeds . . . . .	392
Nevitt, John George, M.R.C.S., Chapel Allerton . . . . .	393
And others omitted for want of space.*	

302.—I have seen no such case. Have seen husband and wife both consumptive, and die from phthisis, both being members of families suffering from chest disease. Examples: Two brothers married sisters; elder brother died of phthisis, wife survives, with cough; children died consumptive. Second brother and sister died of phthisis (husband and wife). Parents of both long suffered with winter cough, lived to old age.

303.—I am not aware that I have seen a case of phthisis directly communicated from one person to another without some hereditary predisposition to the disease, or family relationship between individuals concerned.

304.—I have no recollection of any particular case caught by contagion at present. Phthisical diseases have during the course of my service as military surgeon much diminished in barracks at home, since increased cubic space, ventilation, and light were given to barrack rooms.

Date.		Admissions.	Deaths.	Invalid.
1860.	Tubercular . . . . .	17·82	3·47	5·14
	Respiratory . . . . .	106·48	1·77	2·42
1880.	Tubercular . . . . .	11·1	1·98	4·15
	Respiratory . . . . .	75·7	1·37	1·35

305.—I know a case where a gentleman slept with his wife up to her death of pulmonary phthisis, her illness lasting about six years, during which time she was twice confined. He remained well during the time, and is now enjoying good health. This case was from 1850 to 1856.

306.—Have observed several instances of husband and wife both suffering from phthisis, but have had no direct proof that the one received it from the other.

307.—I have never seen such a case, but I have several times seen cases where the husband and wife, both with strong family predisposition to the disease, have died within a few months of each other. One case about a year ago. The husband's father and mother both died of phthisis, and the wife's mother died of the same disease.

\* See note on p. 85.

309.—The following six cases selected from a list of eighty-eight deaths from phthisis occurring in my practice during the last twenty years, from 1861 to 1882, may possibly be useful to the Committee:—

- |         |   |  |
|---------|---|--|
| Case 1. | { | Husband, <i>æt.</i> 42, beerhouse-keeper, died during 1859. No family history.   |
|         | { | Wife, <i>æt.</i> 40, died during 1861. Her brother died of phthisis.   |
| Case 2. | { | Wife, <i>æt.</i> 28, died 1863. No family history.   |
|         | { | Husband, <i>æt.</i> 36, died January 6, 1869. A tanner.  |
| Case 3. | { | Wife, <i>æt.</i> 47, died December 11, 1864. No family history.  |
|         | { | Husband, <i>æt.</i> 54, died July, 1873. A labourer.   |
| Case 4. | { | Wife, <i>æt.</i> 29, died June, 1873. No history.  |
|         | { | Husband, <i>æt.</i> 40, died 1881. Boiler inspector.   |
| Case 5. | { | Wife, <i>æt.</i> 40, died 1870.  |
|         | { | Husband, <i>æt.</i> 50, died September, 1872. Butcher.   |
| Case 6. | { | Daughter. A married daughter, deserted by her husband, living with her mother, and nursed by her during her illness, died in winter of 1874. |
|         | { | Mother. The mother, 53, died March, 1881.  |

I give these facts without venturing on an opinion upon them. As the result of experience, I may state that I do not think that the contagion theory is tenable, and that it will require much practical investigation before it is accepted generally. Permit me to trouble you with my personal experience *per contra*. My late partner died of phthisis at 50, in 1861. During his last illness, which lasted over a period of some twelve months, I occupied the same bedroom and attended to his requirements. At the same time my professional work was very fatiguing. However, I am at present nearly 50, and never have shown the slightest tendency to pulmonary disease, although in early life I suffered from strumous disease of the knee joint.

311.—Like everyone else, I have seen cases of two, three, and more of a family, generally sisters, suffering one after the other, but that does not enable me to answer the question in the affirmative.

In a subsequent note Dr. Church adds, "I had in my mind two families, in both cases of those who lived to adult life, the sisters living unmarried together, and under the same circumstances, two in the first family and four in the second became consumptive: there being no known predisposition to phthisis in either family. In one family the brothers are now alive and well, and well on in years. In the other family (the one in which four sisters became consumptive) a brother died in early life from hip-joint disease, and one of the sisters also had hip-joint disease. The remaining brother is an unusually active and robust man of 70. The mother of the second family lived to 84. The father died young from some acute disease. The father of the first family killed by a fall from his horse. The mother (who was the only one of seven brothers and sisters who died under 70) died within a year.

315.—This is a question which has interested me for a very long time past. I have closely watched all the cases which have come under my care, and have endeavoured to sift the evidence which has been from time to time put forth in the medical journals. Since I have been in practice here I have seen a very great deal of phthisis, and have come to the conclusion that what might have been called contagion has been a case where one cause has produced the same effect on many people living in the same conditions. For instance: two sisters sleeping in the same bed, and other members of the family in different parts of the same room, have all been more or less phthisical, and one sister has recently died. Has she infected the others or have they not all been infected from one common cause? In the case of husband and wife I have never yet seen anything to arouse a suspicion that one has infected the other.

316.—I have had two cases of chronic phthisis in men, Q. G. and A. B., under my observation for some time, one for six years, who constantly sleep with their wives. Neither of the wives has shown symptoms of phthisis, though in one case (that of A. B.) the wife was always delicate-looking, and is still young. She has slept with her husband during periods of acute exacerbation of the disease.



I know of another case, of a man, D. H., who slept with his wife during a long illness from phthisis, of which he died in 1881. The wife has always been and remains in good health.

317.—After a large experience and long observation I have failed to trace any case of consumption to direct contagion. Cases of wives worn out by nursing consumptive husbands, and becoming themselves consumptive, I have many times noted, but in no case could I put the disease down to contagion exclusively.

320.—I have not seen such a case, but I have observed in a recent number of the "Journal" that it was stated of the late Dr. Cotton that he did not hold the doctrine of the communicability of phthisis pulmonalis. When attending a patient with me the year before he died, he decidedly expressed an opposite opinion. The patient, a girl, the same age as one of my daughters, and a dear friend of hers, had scrofulous parents, and Dr. Cotton advised me on no account to permit my own child to visit her dying friend.

329.—I beg to refer to what I consider a most striking example of the non-contagiousness of this disease which has come under my personal observation. The husband, a tall and, previous to his illness, healthy-looking man, whose father had died of phthisis, his brothers having also a very sickly appearance, married a healthy young woman of good family history, no consumption ever being known to exist on either of her parents' side. Twelve months after marriage she gave birth to a male child, which died, aged  $3\frac{1}{2}$ , of tuberculosis. Four years after marriage the husband's health broke down, and pulmonary phthisis manifested itself, with strumous disease of the bones of the foot. The wife attended him most assiduously, regularly slept in the same bed with him, her own dress being night after night bathed with his perspiration. Had three children by him during his illness. Although exposed to the risk of infection and contagion through child-bearing, constant exposure, &c., she still retains her vigorous health, the husband having died two years ago.

339.—I have known cases occur one after another, at considerable intervals of time, in members of the same family, who have lived in the same house under the same unsanitary conditions of soil, drainage, and ventilation, with hereditary taint or *inborn constitutional tendency—perhaps as a result of the amalgamation of incompatible elements of temperament, &c., in the parents.*

340.—The subject is important. In all my experience I have not met with a single case which I could attribute to contagion. But I have met with many in apparently sound constitutions, induced by daily and prolonged confinement in very limited and ill-ventilated apartments. I am certain that confinement in an atmosphere contaminated with respired air, neglect of exercise and of personal cleanliness, if persisted in for five or six months, will induce the disease in the majority of individuals.

350.—(1.) Mother and daughter living together. Mother died last week. Daughter apparently unaffected.

(2.) Man, wife, daughter, and four sons living together. All four sons died in the last three years; father, mother, and sister apparently unaffected.

(3.) Two sisters living together. One now ill, the other healthy.

Cases of phthisis very common in this neighbourhood. None noticed where contagion could be proved.

392.—Although my attention has been given for many years to this question, I never have observed a case of even probable transmission of phthisis from one person to another.

393.—I have *never had two cases in the same family*, although the conditions were in some instances extremely favourable to such result—*e.g.*, living in the same room and sleeping in the same bed.

Answers coming in this class have been received also from:—

E. G. Pitt, M.D., St. George's East, E.C.; G.M. Bacon, M.D., Fulbourne; Edwin Slade King, M.D., Ilfracombe; D. M. Williams, L.K.Q.C.P., Liverpool; R. Hogarth Clay, M.D., Plymouth; T. Frederick Pearse, M.D., Haslemere; W. B. Cheadle, M.D., Hyde Park Place, W.; William H. Lush, M.R.C.S., Market Lavington; Carey Coombs, M.D., Castle Cary; Richard Neale, M.D., Boundary Road, N.W.; Edward Baker Stephens, L.F.P.S., Doddington; Henry Sutherland, M.D., Whitehall, S.W.; Thomas Webster, L.R.C.P., Redland; Edward Casey, M.D., Windsor; H. Fearnside, M.B., F.R.C.P., Rome; T. M. Lowndes, M.D., Egham Hill; T. A. C. Macarthur, L.R.C.S., Southwold; A. B. R. Myers, M.R.C.S., Surg. Coldstream Guards, Caterham; William Goldie Stevens, L.R.C.P., Renfrew, N.B.; Ed. Fowler Scungal, M.B., New Mill; J. Bisshopp, L.R.C.P., Tunbridge Wells; William White, M.D., Hadfield; H. Hope, L.R.C.P., Southampton; W. Douglas Powell, M.D., Wimpole Street, W.; T. Hall Redwood, M.D., Rhymney; Robert W. Williams, L.R.C.P., Stoke; W. Scott, M.D., Dublin; J. Roberts Thomson, M.D., F.R.C.P., Bournemouth; Henry F. Marley, L.R.C.P., Padstow; F. W. O'Connor, F.R.C.S., Limerick; Alfred Thomas Brett, M.D., Watford; William Murray, M.D., Burley-in-Wharfedale; William H. Brace, M.D., Queen's Gate Terrace, W.; George Cordwent, M.D., Milverton; E. J. Davies, M.B., Liverpool; W. H. Day, L.R.C.P., Pentonville, N.; H. M. Duncan, M.D., South Hampstead, N.W.; T. M. S. Foggo, L.K.Q.C.P., Kensington, W.; John A. Gill, M.D., Stratford-on-Avon; Frank S. Goulder, L.R.C.P., Dudley; Charles F. Hutchinson, M.D., Scarborough; C. Handfield Jones, M.B., Montagne Square, W.; Bernard Kelly, M.D., Rotherhithe, S.E.; R. M. Miller, M.D., Upper Norwood, S.E.; William Hartigan, M.K.Q.C.P., Hong Kong; Richard Wood, M.D., Bromsgrove; Richard Jackson, M.R.C.S., Surg. Maj. A.M.D., Paclamarhi, India; C. L. H. Pemberton, M.R.C.S., Banbury; T. R. H. Chum, M.R.C.S., Prestwich; W. R. S. Jefferiss, M.D., Burton-on-Trent; Abraham Kidd, M.D., Ballymena; Abraham Colles, M.D., Wellington; George Thornton Mockett, L.R.C.P., Tyldesley; John O'Dowd, L.F., P.S., Dudley; Edward Gordon Hull, M.D., Stockton-on-Tees; James Crawford, L.K.Q.C.P., Ightham; William Hooper Masters, M.D., Thrapstone; James S. Laing, M.B., Skene, N.B.; Benjamin Clarke, F.R.C.S., Upper Clapton; H. Strangways Hounsell, M.D., M.R.C.P., Torquay; Fred. H. Haynes, M.D., M.R.C.P., Leamington; Robert Barrington Cooke, L.R.C.P., F.R.C.S., Scarborough; C. Taylor Brown, M.B., C.M., Maryfield, N.B.; William M. A. Wright, M.B., Dalkey; J. Cross Johnstone, M.D., Brig.-Surg. A.M.D., Bedford; John Williams, M.D., Ventnor, Isle of Wight; A. Hirst, M.R.C.P., Prestwich; C. Mason Scott, F.R.C.S., Rockingham; Charles E. Shelly, M.B., Hertford; A. C. Rayner, M.D., Preston; John Patterson, M.D., Constantinople; James Crocker, M.R.C.S., Bingley; John Dale, M.R.C.S., Stockton-on-Tees; James Alexander, M.D., Paynton; Francis Benjamin Brodribb, M.R.C.S., Colne; William Kitto Giddings, M.R.C.P., Calverley; Thomas Smailes, L.R.C.P., Honley; T. Wells Hubbard, M.R.C.S., Bromley; J. Bain Simecock, M.R.C.S., Bridgwater; R. Gillard, M.R.C.S., Clapham Road, S.W.; James McNaught, M.D., Boothfold; John Hern, M.B., Darlington; S. Wellesley Coombs, F.R.C.S., Worcester; W. A. Bonney, M.D., Chelsea, S.W.; A. Cummings Air, L.R.C.P., Kennington Park Road, S.E.; D. Biddle, M.R.C.S., Kingston-on-Thames; William C. Blackett, M.R.C.S., Durham.

## A PRELIMINARY REPORT ON ACUTE PNEUMONIA.

MORE than 400 reports have been received by the Committee, and 350 of these have been analysed with reference to questions of etiology, epidemic prevalence, communicability and other points mentioned in the Memorandum upon this subject. Acting, however, under the instructions we have received, the publication of the Tables that have been drawn up, together with a detailed synopsis of results, is deferred until further reports have been received and tabulated. Much of what follows must be regarded, therefore, in the light of provisional or tentative conclusions which await confirmation or refutation in the future when the number of observations is much larger.

Following the scheme of the Memorandum and of the cards issued, the general results of our analysis of 350 cases may be conveniently arranged under the following headings :

1. The apparent influence of sex, age, habits, &c.
2. Sanitary and meteorological conditions respectively in their etiological relations.
3. The acute specific fevers and infectious diseases apt to prevail coincidently with the prevalence of pneumonia.
4. The morbid associations of pneumonia in so far as these appear by reference to (1) the family history, and (2) the personal history.
5. The symptoms and modes of onset of individual cases, with reference especially to (1) those with and those without premonitory symptoms, as well as to (2) the proportion of cases exhibiting gastro-intestinal symptoms and suggestive of pythogenic origin.
6. The comparative frequency of pneumonia in the several regions of the lung respectively ; the favourite days of crisis ; and the proportion of examples ending suddenly in that way against those that terminate by gradual subsidence.



7. Evidence bearing upon the question of the asserted communicability of pneumonia from one individual to another.
8. The nature and origin of so-called epidemic pneumonia.
9. The prevalent methods of treatment in this country.

### 1. *Sex, Age, and Mortality.*

In an enquiry such as this too much importance may easily be assigned to the statistical results as regards the prevalence of pneumonia amongst the sexes, or at different periods of life; for the cases have been gathered indiscriminately from all parts of the country, and of necessity include but a small proportion of all that have occurred within the time over which the returns extend. This report would, however, be incomplete if it did not make mention of these points, which possess a certain relative, if not an absolute value, particularly when dealt with in reference to the mortality from the disease.

The total number of cases analysed at present amount, as stated, to 350. Of these 229 were males, 120 females, the sex not being stated in one case. Thus, roughly speaking, the proportion of males to females is nearly 2:1.

As regards age, we find two cases given of infants under one year; from that age to ten years there are 43 cases. Between the ages of ten years and sixty years there fall 269 cases, of which number 195 occurred between ten and forty years, the greatest prevalence being between ten and twenty and twenty and thirty, each of these decades yielding 70 cases. Nine cases are given between seventy and eighty years, and one over eighty, who recovered. In 7 cases no age is stated.

Passing now to the question of mortality (the main test of the severity of the attacks), it is convenient to consider it from various points of view—so as to ascertain, if possible, particular external or internal conditions influencing the course and termination of the disease. These conditions are dealt with more generally in other parts of the report, and they will be discussed here solely with reference to mortality. They are: (*a*) sex and age; (*b*) habits and mode of life; (*c*) sanitary conditions of the dwelling; (*d*) the type of the pneumonia as shown by its mode of onset; and (*e*) the extent of the pulmonary inflammation.

Of the 350 cases, 282 recovered and 68 died, giving a percentage mortality of 19·4.

(a.) Of the 229 males 43 died; mortality = 18·7 per cent.

Of the 120 females 25 died; mortality = 20·8 per cent.

The influence of age upon mortality in pneumonia can only be estimated by a much larger collection of cases than the present. So far as these cases go, we find the rate of mortality rising with each decade of life from ten years to forty. Thus: Under ten years of age there were 45 cases, 3 deaths; between ten and twenty, 70 cases, 5 deaths, mortality 7·1 per cent.; between twenty and thirty, 70 cases, 8 deaths, mortality 11·4 per cent.; between thirty and forty, 55 cases, 10 deaths, mortality 18·1 per cent. These rates are below the general average noted above; but in the next decade—forty to fifty—42 cases with 17 deaths, the mortality is more than doubled, viz., 40·4 per cent.; between fifty and sixty there were 32 cases with 11 deaths, mortality 34·3 per cent. Beyond sixty the number is too few to infer anything as to the mortality rate; but we may state that between sixty and seventy there are 19 cases, 3 deaths, and from seventy years and upwards 10 cases and 6 deaths; one patient, who recovered, being over eighty years of age. The age has not been given in seven cases, two of which were fatal.

(b.) As to habits and mode of life, we find that 228 are recorded as “temperate,” 80 as “total abstainers,” and 37 as “intemperate,” no statement being made on this head in five cases. In only 30 cases is the food stated to have been insufficient.

				Per cent.
Temperate	.	186 recovered.	42 died.	mortality 18·4
Total abstainers	.	71 „	9 „	„ 11·2
Intemperate	.	22 „	15 „	„ 40·5
Not stated	.	3 „	2 „	
Food, sufficient	.	260 „	60 „	„ 18·7
„ insufficient.	.	22 „	8 „	„ 26·6

If any conclusion may be justifiably drawn from these figures, it is that whereas individuals of temperate habits and those who take a sufficiency of food incur about an equal liability to fatal

pneumonia, the total abstainers enjoy a far greater immunity ; whilst amongst the intemperate the death rate is very high indeed. The figures are, however, too few to permit of such a deduction except, perhaps, in the case of the intemperate, where the result is in accord with general experience. An insufficient supply of food, which in many cases goes along with alcoholic excess, does not, according to these results, seem to be so fatal a determining factor as the latter.

(c.) Thirty-six fatal cases came from houses of which the sanitary condition is reported as "good," and 31 where it is noted as being "indifferent," or "bad." In one it is "doubtful." It will be seen further on that this proportion corresponds very nearly with the general proportion of cases occurring under these conditions respectively ; so that we are unable to assert from these returns that insanitary surroundings form a main factor in the occurrence of a *fatal* type of pneumonia.

(d.) But is there any evidence to show that the type of pneumonia attended with a fatal issue differs from that of non-fatal cases ? Of the 68 fatal cases, 34, or precisely one-half, were perfectly "typical," commencing suddenly with pain and rigors ; the rest arose more gradually, with premonitory symptoms lasting for a few days, six of these being preceded by catarrh. Thirteen of the fatal cases exhibited gastrointestinal symptoms either before the onset of the illness or during the attack ; but in one-half of this number the symptoms in question consisted merely in vomiting (v. § 5).

(e.) Of the 68 fatal cases, one lung alone was involved in 37, viz. :—

Right side—Apex, 1.	Base, 17.	Apex and base, 4.
Left side — „ 2.	„ 10.	„ „ 3.

Both lungs were attacked in 31 cases, the signs being limited to the apices in one case, to the bases in 22. The whole of both lungs " was affected in 2 cases ; the



whole of the left lung and the right apex in 2; left lung and right base in 2; and right lung and left base in 2.

## 2. *Sanitary and Meteorological Conditions.*

The sanitary condition of the houses in which the patients lived was reported "indifferent" or "bad" in 170 out of 342 cases, and "doubtful" in 8. Of 341 districts where pneumonia was said to be prevalent, 168 were "indifferent" or "bad" in sanitary respects, and 173 good. It thus appears that the sanitary condition of nearly half the houses is unsatisfactory, and in only 122 instances out of the 350 (hardly more than a third) is the sanitary state "good" as regards both house and district.

Although it would thus appear that insanitary conditions play a not inconsiderable part in the etiology of pneumonia, it is impossible to question the fact that marked influence is exercised by special meteorological states. Without venturing to assert in what this influence precisely consists, we think that few would deny that the state of the atmosphere as regards barometric pressure, humidity, temperature, and direction of the wind, does operate in exciting or favouring the development of this disease. This, indeed, is the general opinion in this country, and amongst the returns submitted to us are to be found several in which the attack is distinctly attributed to exposure in inclement weather. Exposure, then, is to be admitted as a not infrequent cause, but largely in association with such disposing conditions as bodily fatigue, exhaustion, and subjection to insanitary conditions. This question is a most difficult one, not perhaps to be determined by statistical results, or by such facts as can alone be gathered from an enquiry like this.\*

\* As bearing upon the influence of seasonal and meteorological conditions, Dr. Longstaffe has compiled some valuable tables from the Registrar General's Reports of the comparative mortality from pneumonia and bronchitis in London; and has placed them at the disposal of the Committee. We trust they will find a place in our final report, when we shall hope to deal more exhaustively with this part of the enquiry. Meanwhile it suffices to state that Dr. Longstaffe's tables show a rather striking parallelism in the rates of mortality from these two diseases, with this notable difference, that the death rate of bronchitis appears to be proportionately more influenced by exceptional atmospheric conditions—(e.g., the fog in February 1880), than is that from pneumonia.

Reserving the full analysis of the weather conditions registered in these 350 cases, we may state in general terms that more than one-half of the total number of cases arose when the weather was damp or wet, and even a larger number when it was cold, the combination of cold and wet being particularly favourable for the development of the disease. It is not surprising, therefore, that the most prevalent winds at the time of attack should be N.E., E., and S.W.—the last predominating, perhaps, through the accident of its more frequent occurrence in this country.

### 3. *Specific infectious Diseases prevalent along with Pneumonia.*

The result of our enquiry as to the several affections, specific or infectious, which are apt to prevail, coincidently with pneumonia,\* tends to negative the belief in any direct or intimate connection with these conditions.

The occurrence of *enteric fever* at the same time and in the same house with pneumonia was noted only in a single case, and in only 29 instances was enteric fever known to prevail in the same district. Similarly with *diphtheria*. It is rare to meet with it in connection with that acute primary pneumonia to which alone the enquiry refers. The case is somewhat different with *erysipelas*. In 66 instances of the 350 erysipelas prevailed in the same district. Moreover, the instances of pneumonia occurring in this connection are chiefly examples of that typical form of the affection which has an orderly course and ends by sudden crisis. If such typical cases were to be selected from the rest this same number, 66, would still represent the proportion of instances of prevalent erysipelas out of a much smaller number than 350. It is to be mentioned at the same time that pneumonia and erysipelas are not found in actual juxtaposition, prevailing together in the same house and at the same time.

The nearest and most obvious connection of pneumonia with acute inflammation of whatever kind is that with *tonsillitis*, which is mentioned as prevalent in 105 instances, or, excluding the 31, which are defective on this point, in nearly a third. *Herpes*, on the other hand, prevailed in but 24. But it is mani-

\* In thirty-one instances no information is given under this heading.

fest that an affection of this trivial kind must often escape professional observation. The concurrence of pneumonia and herpes is a fact already sufficiently established. Whether or not this particular eruption is unduly prevalent along with the prevalence of pneumonia, our information does not enable us to determine.

#### 4. *Family and Personal Histories.*

From this review of the affections occurring at the same time as pneumonia we go on to enquire as to the diseases associated with it in *the family or personal histories of the patients*. Do individuals who become subjects of pneumonia exhibit in excess any particular morbid predisposition? Is the liability to this disease transmitted, or does the fact of having suffered pneumonia render the individual more susceptible than another? So far as our material teaches, these questions (perhaps with the exception of the last) must be answered in the negative. Thus, grouping together the several lung diseases included in the term phthisis, there is a phthisical family history in but 34 of the 350 cases under review, a number not exceeding—probably falling below—the proportion of phthisis which would appear in a like number of our Anglo-Saxon race taken at hazard.

It may be here remarked that in the examples of pneumonia now before us there is a singular absence of individuals belonging to families in which the tubercular inheritance is prominent and well marked.\*

Of the *sequelæ* of pneumonia it may be said generally that the common rule is complete recovery. Five cases are reported as leaving behind some signs of consolidation; 3 are said to have ended in “chronic pneumonia” or “phthisis;” and in one case suspicious physical signs persisted at one apex; “pleurisy” continued in 9; 3 led to empyema, and 1 to fatal pneumo-hydrothorax. In a very few cases mention is made of ensuing debility with slight cough.

\* The number of instances where pneumonia finds mention in the *family history* is very small, probably not exceeding  $3\frac{1}{2}$  per cent.



As to the *liability to recurrence* in pneumonia there are 30 such cases in 350. Of other antecedent diseases none is especially prominent, and acute rheumatism in that relation was noted but 6 times.

#### 5. *Modes of Onset.*

In an attempt to differentiate cases of pneumonia, much valuable information may be expected from facts concerning *the mode of onset of the illness*. A certain proportion of cases begin suddenly, without any premonitory symptoms, the rigor occurring as the first indication of departure from health. In several such cases there is a distinct history of exposure to cold, wet, or fatigue within a few hours of the seizure. Again there is a considerable class—of which the returns before us give numerous examples—where for a few days preceding the actual onset, malaise, or weakness, muscular pains, headache, anorexia occur; or a slight cough or catarrhal symptoms, bronchitic mainly. Or, lastly, there is in some a predeminance in this premonitory stage of more or less gastro-intestinal derangement, as evidenced by nausea, vomiting, or diarrhœa. In a few cases such symptoms occur simultaneously with the rigor and pneumonic “stitch”; but in a larger number some days elapse before the disease declares itself unmistakably as pneumonia. Sometimes the onset is so gradual and insidious—or, more strictly speaking, these premonitory symptoms endure so long—that a diagnosis of typhoid fever is made, only to be dispelled when the occurrence of local pain and rigor, and the definite signs of pneumonia point to the real nature of the case. Such differences in the mode of onset are noteworthy, for they may assist in clearing the ground for a separation of the disease into distinct types. At present we must content ourselves with a numerical analysis of these varieties:—

Premonitory symptoms were present in about 270 cases.			
“	“	absent in about	80 “
Rigor occurred at the onset in	.	.	241 “
“ was not present	“	.	39 “
Catarrhal symptoms preceded the attack in			40 “
Cough	“	“	29 “

Headache symptoms preceded the attack in	53 cases.
Malaise                   ,,                   ,,                   ,,	49   ,,
Erysipelas           ,,                   ,,                   ,,	1   ,,

The total number of cases in which *gastro-intestinal* symptoms were present was 59,—in 7 of these after the onset of the pneumonia. Of these symptoms, nausea and vomiting occurred in 35,—in 21 of which a rigor ushered in the declared symptoms. Diarrhœa was the only symptom in 13 cases (in one, No. 71, with hæmorrhage),—a rigor occurring in 8. Vomiting and diarrhœa existed together in but 3 cases, in one of which there was a rigor. Thirteen of these cases were fatal, or 22 per cent. of the number so attacked, a figure somewhat above the average mortality.

#### 6. *Seat of Lung Inflammation, Days of Crisis, &c.*

(a) The *situation of the lung inflammation* is a point of interest in reference to the proportion of cases of double pneumonia, and of so-called apex pneumonia; as well as to the comparative frequency with which this or that locality of the lung becomes the seat of attack. The general belief in the greater frequency of *right* pneumonia is here borne out by 108 cases in which the right base was affected against 86 of the left base; and 16 of the right apex against 10 of the left. That the inflammation should have been confined to one or other apex in but 26 cases out of the whole number is remarkable. We venture to think (and are supported in this belief by the present returns), that “apex pneumonia” is apt to occur in groups at certain times and seasons, and that ultimately the above proportion will be found to be below the average.

In one instance, a fatal case, verified by post-mortem, *both apices* are affected, a solitary example which calls attention to the extreme rarity of pneumonia of this seat, and contrasts strikingly with 76 cases where both bases are involved. Next in rarity is the implication of both bases along with one apex. This combination occurs in but 5 cases as regards the left apex, and in but 3 as regards the right apex. By far the commonest seat is the right base.

In 334 cases the numbers are :—

Right base . . . . .	108
Left base . . . . .	86
Both bases . . . . .	76
Right apex . . . . .	16
Left apex . . . . .	10
Right base and apex . . . . .	12
Left base and apex . . . . .	8
Both bases and right apex . . . . .	3
Both bases and left apex . . . . .	5
Whole of right . . . . .	3
Whole of left . . . . .	4
“ Whole of both lungs ” . . . . .	2
Middle lobe . . . . .	1
	<hr/>
	334

(b) On comparing the number of cases *ending suddenly with those that had a gradual subsidence* we find that out of 306 (excluding doubtful and a proportion of fatal cases), there are 138 of the former to 168 of the latter. In other words the crisis of acute pneumonia, to which many attach importance as among the indications of the specific character of the pneumonic fever, is more often absent than present. It will be asked whether the cases terminating in crisis are in other respects separable from those that terminate gradually. To this enquiry, however, we are not as yet prepared with a reply. As regards the seat of inflammation, the suddenness or otherwise of attack, and its apparent origin in the two cases respectively, there is no obvious or well-marked distinction to be made.\*

(c) With reference to the duration of pneumonic fever it is matter of common knowledge that *the days of crisis* commonly fall somewhere between the third and the eighth day. Of these days the seventh is the favourite, and the sixth comes next, leaving the eighth day far behind. Thus, out of 290 cases, 65 had their

\* Gradual subsidence appears upon our figures somewhat more common with single than with double pneumonia.



crisis on the seventh, and 43 on the sixth day,\* while the number ending in crisis on the eighth day are but 29, less than half those on the seventh. Crisis later than the eighth day is very exceptional. It is with us more frequent on the tenth than on the ninth day (29 to 19). There are but 7 instances of crisis on the eleventh day, and but 2 at any later period.

### 7. *Communicability of Pneumonia.*

As regards the *conveyance of pneumonia from person to person*, 12 reports reach us which are supposed to be illustrations of such conveyance. The most striking cases are those by Dr. Darwin, of Didsbury (98), where the husband, it was believed, took the infection from the wife; by Dr. J. Bury, of Manchester (99), where a child, not subjected to exposure or insanitary influence, seemed to take pneumonia from its companion; by Mr. Bowen, of Liverpool (159), where four members of a family were attacked in succession without known exposure, and where the sanitary state was "good" both in house and district; and of Dr. Lane, of Bishop's Castle (204), where the wife was believed to have taken pneumonia from her husband. "I have no doubt," writes this observer, "that the patient's attack was due to direct infection, she having never left her husband's room except for a few moments from the time of his seizure until his death."

The other instances of supposed communication† are less pertinent than these, owing to circumstances, sanitary or meteorological, which make it more or less probable that the successive attacks were due to some common cause. Such, however, is not the interpretation of those best qualified to judge, namely, the observers themselves. In our detailed Report all the cases of supposed direct communication will be fully stated, including some of which we have notice, but which are yet to be received.

\* In 290 crisis fell between 3rd and 14th day, and the distribution between 3rd and 8th is as follows:—3rd day, 10; 4th, 25; 5th, 30; 6th, 43; 7th, 65; 8th, 29. With this compare 324 quoted by Dr. Wilson Fox out of a total of 374 (Reynold's System, iii., p. 650), where the 5th and the 7th are the favourite days: 3rd day, 31; 4th, 33; 5th, 78; 6th, 50; 7th, 96; 8th, 36.

† Numbered respectively 11, 12, 123, 152, 166, 207, 240, 260, 335, 336.

The reader will be then in a position to judge of the value of this very important head of evidence.

### 8. *Epidemic Pneumonia.*

In considering the question of so-called *epidemic pneumonia*, we have been careful to distinguish between examples where the affection becomes prevalent, owing to meteorological, rather than "septic" causes. No one doubts that, like bronchial catarrh, it is largely influenced by weather; and we have already adduced (*vide* § 2) some evidence to that effect. The significance, therefore, of pneumonia occurring in groups is ambiguous; and each group must undergo separate scrutiny. In our 350 cases we find 46 where there were other cases of pneumonia (one or more) in the same house; and 96 where there were other cases in the same district. In most of these, however, catarrhal affections prevailed as well. Yet even so, such examples of pneumonia occurring *in groups* are quite exceptional. Only four such instances have in fact been reported to us. Two of the epidemics occurred some years ago—the one at Holmwood, near Dorking, an account of which was given by the Medical Officer of Health, Mr. E. L. Jacob, in his Annual Report for 1877; the other at Long Handborough, near Oxford, for the details of which we are indebted to Dr. Gilbert Child, of that city, he being Medical Officer of Health for the district at the time of the epidemic (1878). The third instance is mentioned by Dr. White, of Manchester; and the fourth, an interesting account of an outbreak in a school boarding-house, is furnished by Dr. Gooch, of Eton. It will suffice here to transcribe these accounts, in the hope that further definite information upon epidemics as they arise will be supplied to the Committee.\*

1. Epidemic of Pneumonia at Holmwood, Surrey, December, 1876, to June, 1877. (From the Annual Report of the Medical Officer of Health, Mr. E. L. Jacob.)

"During the first half of the year, there was an epidemic of *Pneumonia* on Holmwood Common. The first case occurred in the previous December (1876),

---

\* It is proposed to deal more fully with this subject in a future contribution to the Record—by the collation of all recorded instances of the kind.

and during the following six months, eighteen persons, living in twelve houses, were attacked with it. Two of the houses were invaded in December, one in February, two in March, two in April, and five in May. In two instances there were three cases in each house; and in two other instances there were two cases in each house, while the remaining eight houses had only one case each. At two houses the second case appeared within a few days after the first, and at another it appeared a month after the first. At the fourth house, the disease re-appeared three months afterwards. Eight of the patients were men, and ten were children, the women escaping. Five were removed to the Cottage Hospital at Dorking, and three others (two of whom were men) died. All of the infected houses were cottages, and all were situate in the immediate neighbourhood of the Norfolk Arms Inn at North Holmwood. Seven of them formed part of a group of twelve, called Peters' Cottages, and the rest were detached buildings. The total number of houses in that part of the Common is about thirty. The disease did not appear to be in any case a secondary affection, such as supervenes in the course of typhoid fever and other similar diseases.

"None of the illnesses were attributed to exposure to cold or wet, but the site of the houses is decidedly damp during the greater portion of the year; soil, clay. The "general sanitary arrangements" of the premises were not found to be bad, and no particular water-supply, milk-supply, or sewer appeared to be responsible for the origin or spread of the disease. There was also little or no evidence that the disease had spread from house to house by contagion."

## 2. Epidemic of Pneumonia at Long Handborough, Oxon, April, 1878. (From the unpublished Report of the Medical Officer of Health, Dr. Gilbert Child.)

"A remarkable outbreak of a disease not usually recognised as belonging to the zymotic class occurred at Long Handborough in the month of April. This was an epidemic of pneumonia, which carried off no less than eight persons in this village between April 6th and April 27. In all cases the victims were adults—the youngest 22 years of age, the oldest 87, and the remaining six between the ages of 38 and 60; and in every case the progress of the disease was rapid, death occurring in most cases within a week of the attack. One remarkable circumstance in connection with the outbreak was that a woman living at Witney (about five miles distant), a near relation of one of the persons who died at Handborough, walked over to the latter place to attend the funeral, remained there for the night, sleeping in the bed from which the deceased person had been removed. She returned to Witney on the following day, sickened with the same disease, and died within a few days. This might seem to suggest that the complaint was infectious, but the conclusion is by no means certain, as it is clear that if the disease depended on any local conditions, the visitor had in this case subjected herself, for the time, to almost the precise combination of them which prevailed more permanently among the inhabitants of Handborough. At the same time I must admit that I am quite unable to assign any local conditions as the cause of this outbreak. A few similar ones have been recorded elsewhere, amongst which I may mention one at Holmwood, in Surrey, about a year before the present one; another of which I have been informed at a large school in Berkshire several years ago; and one at Cowley, near Oxford, almost simultaneously with that on which I am now reporting. At present I do not know of any satisfactory explanation which has been suggested for such outbreaks."



3. In connection with a case (No. 313) reported by him, Dr. White, of Hadfield, Manchester, states that—

“An extensive epidemic of pneumonia occurred in the neighbourhood some months ago.”

In reply to further questions he writes (May 28, 1883) regretting his inability to give detailed information. There was no instance in which more than one person in the same house had the disease, and no suspicion at all of contagion. The cases were in persons of all ages—mostly children; and most of them recovered. The neighbourhood is hilly, cold, damp and exposed; and the drainage is moderately good. “The inhabitants are well-to-do cotton operatives, and live in good houses, in large villages, and are by no means overcrowded.”

4. Dr. Gooch, of Eton, writes (June 5, 1883):—

“The cases, the details of which you wished me to forward, were very interesting. . . . The first case of illness was that of the butler, who, at the beginning of the sick time, was taken ill with acute double pneumonia, and nearly died. A few days after two boys had bad sore throats; and another lad an attack of pleuro-pneumonia, terminating with effusion, which subsequently became absorbed. Two days after, several other cases of sore throat, and two or three of diarrhoea occurred in the house, and then another boy was seized with acute double pneumonia, which ran a severe course. At the same time another boy in another boarding-house was attacked with pneumonia, but he was constantly at lessons in the house first mentioned. All the cases recovered, but there were twenty inmates of the house more or less ill during a period of about three weeks.

“We examined the water and milk supply, also the drainage, which was imagined to have been in perfect order, and we ultimately discovered that the trap from the sink in the butler’s pantry was defective, and through it sewer gases were escaping into the house. In this pantry the butler slept, and the pupils’ room and boys’ rooms are not far distant from it. I think there can be no doubt but that all this illness was caused by the one defect in the drain mentioned, for there was no epidemic of scarlet fever or any other illness at the time in the neighbourhood.”

## 9. *Treatment.*

A glance through these returns brings strikingly into view the great diversity that exists in the *treatment of acute pneumonia*, and at the same time enforces the fact that the disease runs a definite course, no matter what line of treatment be adopted. It is hardly to be expected that any analysis of these reports can teach us much as regards the suitability of one rather than of another method; the cases are not dealt with under the rigid restrictions of an experimental inquiry; and their number is far too few to admit of any useful comparison. Moreover, it must be borne in mind that the prescription of alcohol and other

cardiac stimulants, although not to be looked upon as a routine measure, is often imperatively called for; the tendency to death being mainly from cardiac failure. This fact is by itself sufficient to show how valueless any deduction would be from the comparison of the mortality of cases treated with or without stimulants.\* Indeed, of the two chief methods of treatment—often curiously combined—the restorative and stimulant, and the depressant or sedative (*e.g.*, antimonial), the evidence before us shows a striking preference in favour of the former, the number of cases treated on that plan being nearly five times as many as those treated on the latter method.

We have endeavoured to ascertain by the light of these returns the general opinion in this country respecting the method of treatment so strenuously advocated in Germany—viz., the antipyretic; and we find that very many practitioners avail themselves of the use of drugs believed to have this property—at any rate, in the earlier stages of the disease. Thus quinine was prescribed in 61 cases, aconite in 51, digitalis (less perhaps as an antipyretic than as a cardiac stimulant) in 25. In 14 cases the salicylates were administered.†

Of other remedies the most in favour are saline diaphoretics and diuretics at the outset, followed later by expectorants. Iodide of potassium is given by a few, and calomel is occasionally prescribed. Opiates, seldom long continued, were given in association

\* For instance, it is expressly stated in 16 cases that no stimulants were prescribed, and of these patients one died. In 146 cases (and doubtless in others also, where no mention has been made of it), alcohol in varying forms and quantity was administered; and 53 of these patients died. Not even the most prejudiced advocate of total abstinence would affirm that these results afford any argument against the employment of alcohol in this disease, although the mortality under alcoholic treatment is to that under non-alcoholic as 5 : 1.

† But it is also clear that the measures most in favour with the German school have not taken root here at present. We mean the measures for reducing temperature by the application of cold to the surface of the body. In not one of these 350 cases was the cold bath employed; in only five is it recorded that cold sponging was used; in two mention is made of the wet pack, in two of the application of ice to the head, and in one of the compress. The facts and arguments adduced by Jürgensen and others in support of such measures, embolden us to suggest to our brother practitioners to make—so far as can be done with regard to general condition of the patient—a further trial of some of these anti-pyretic methods, on the ground that the “pneumonic fever” is quite as grave an element in the case as the extent of lung involved in the inflammatory process.

with other drugs in about one-fifth of the cases. Local measures of counter-irritation, poultices, blisters, and leeching are frequently prescribed—often, no doubt, for the relief of the pain due to the concomitant pleurisy. It may be doubted whether such measures have any effect in controlling the inflammation. There is only one case of venesection—a solitary instance of the one measure which, a generation ago, would have been adopted in every case without hesitation.

Lastly, a certain number of practitioners—about as many as those who have prescribed antimonials—state their treatment to have been “expectant” \*—a term which we may understand to include rest, nutriment, possibly some stimulant, and the combating of symptoms as they arise—a method which, recognizing the self-limited character of the disease, abandons any futile attempt to curtail its natural course, whilst maintaining, so far as possible, the strength of the patient.

The foregoing observations will afford some indication of the general scope and tendency of the present inquiry, and may seem in a measure to foreshadow some of its final conclusions. For the present, however, we reserve all comment excepting upon a single point; and to this we allude mainly with the view to elicit further evidence bearing upon that particular question.

Judging from these 350 carefully recorded cases, the material of the present Report, we think the evidence before us insufficient to support the doctrine that pneumonia is a specific fever whose chief local manifestation is in the lung. Like other respiratory diseases, we find it prevailing in certain states of weather, and, apart from all else, the great regulator of its frequency is season. It may be taken for certain that it confers no protection upon the individual, but rather an increased liability to future attacks. It appears to have no direct association with any specific or conveyable disease, and its near alliance with tonsillitis is in striking contrast with its infrequency in connection with diphtheria. Instances of pneumonia undoubtedly occur which are apparently “pythogenic.” But those which have this origin are not other-

\* Only one reporter, Mr. Raven of Broadstairs, uses the term “restorative,” first proposed by the late Dr. Hughes Bennett, one of the earliest in this country to call attention to the natural history of the disease, and its successful treatment without active medication.



wise separable, so far as we see at present, from others which are obviously due to exposure. The sudden termination of the pneumonic fever within a period more or less definite (which occurs, as has been said, in less than half of the cases), suggests a certain likeness to the specific fevers. But here again the examples which thus terminate by crisis are not to be distinguished from those in which the fever subsides gradually, *pari passu* with the recovery of the lung, while it is common knowledge that the cases which exhibit crisis are precisely those that have had their origin in some direct and notable exposure. "Epidemic pneumonia," as judged of by the cases we are now reporting upon (and we are careful to keep out of sight all other evidence), is in part explained by atmospheric conditions and in part by other agencies generally prejudicial to health. If it be admitted, for example, that wherever the tone of health is lowered (so to speak) the occurrence of pneumonia is rendered more probable; and that insanitary surroundings, not less than deficient food or alcoholic excess, favour the development of the disease on that account; many of the so-called epidemics are sufficiently accounted for without invoking any specific agency whatever.

The asserted contagiousness of pneumonia is of course a strong argument on the other side, and calls for careful investigation. But, as has been said, evidence to this effect, at present in our possession, although striking of its kind, is as yet scanty. While, therefore, we would avoid any statement seeming to imply a final opinion upon the material now before us, we feel justified in saying that the evidence as yet collected does not suffice to warrant the conclusion that the so-called acute croupous pneumonia is of specific origin.

It is probable that the collection of 1000 cases will serve to set at rest many of the questions regarding pneumonia which are at present problematical. We have already nearly half that number, and at the present rate of collection professional zeal will have supplied the remainder within a year. With the issue of a final and detailed report the profession will be enabled to judge for themselves as to the bearing of the facts laid before them.

We cannot conclude this preliminary report without expressing on behalf of the Committee our obligations for the manner in

which our appeal has been responded to, the care and accuracy of the individual reports, and the readiness and courtesy with which additional information has been supplied whenever we have had occasion to ask for it.

*The Sub-Committee is indebted for the preparation of this report, to* Drs. OCTAVIUS STURGES and SIDNEY COUPLAND.

(Signed) OCTAVIUS STURGES, M.D.  
T. H. GREEN, M.D.  
J. BURNEY YEO, M.D.  
SIDNEY COUPLAND, M.D.  
F. A. MAHOMED, M.B., *Secretary*  
*to the Committee.*

## A PRELIMINARY REPORT ON CHOREA.

It will doubtless interest those who have contributed to this investigation to learn, from an *interim* report, the general result of the inquiry as far as ascertained, and it will not perhaps be without value to those who are good enough to continue to contribute answers if certain *lacunæ* in the replies are pointed out.

It should be stated at the outset that the character of the replies on the cards offers great encouragement to all interested in collective investigation: they show a precision and conciseness which render analysis comparatively easy, and convey a sense of accuracy that stamps a value on the returns.

The number of filled-up cards now received amounts to over 200, but this report only deals with 128, which have been tabulated. Before the final report is prepared it is earnestly hoped that the cards received will reach a very high number—1,000 are desired—and thus enable many of the questions raised in the inquiry to receive an authoritative, and, perhaps, final answer.

To deal now with the main points of the inquiry. The preponderance of the disease in the female *sex* is well illustrated: there are 90 females to 32 males; in a few cases the sex has not been recorded. *The age incidence* of the disease is in agreement with most statistical inquiries on this point. The great majority of cases occurred between the ages of 8 and 15, in both sexes. Under 5 there was one (female); 5 to 6, three (females); 5 to 7, five (4 females, 1 male); 7 to 8, one (female); 8 to 9, fifteen (8 females, 7 males); 9 to 10, thirteen (9 females, 4 males); 10 to 15, fifty-seven (43 females, 14 males); 15 to 20, twenty-four (18 females, 6 males); 20 to 25, two (females); none between 25 and 40; one each at 43, 78, and 86. These last exceptional cases will be mentioned again later.



*Class in Society.* The great excess of the disease in the lower classes is very marked, and in accordance with general experience: thus in members of the upper classes there are only 3, middle classes 38, lower classes 81. These numbers have only a general value, as allowance must be made for the greater facilities of note-taking in the cases of poorer patients, and the larger proportion of them to the other classes. The more general the inquiry becomes, the more likely any source of error on this point will be avoided. It is hoped that those contributing cards will endeavour to fill them up *in all their cases*, in whatever rank of society they occur, in order that accurate information on this point may be obtained.

*Residence.* No attempt is made on the present occasion to arrange the disease according to geographical distribution. Only large numbers contributed from all parts of Great Britain will enable any conclusions, on this point, to be arrived at. It may be mentioned, however, that the geographical distribution of chorea promises to be one of the most interesting points in the inquiry, and that evidence is already at hand affording unexpected information on the matter. It will be of great interest if those filling up cards in the future will, as some have already done, increase the value of their report by stating *whether the disease is common or rare in their neighbourhood*. As regards *locality*, as far as an inference can be drawn from the cards already examined, this does not appear to have much influence in the production of chorea. It is stated to be high in 54, low in 52, dry in 51, damp in 51, exposed in 50, confined in 32. Insufficiency of food does not appear to take much part in causing the disease: it is only recorded in 14 cases, whilst it is stated that there was sufficiency of food in 106 cases.

*Nutrition.*—Chorea, as is well known, is more apt to occur in persons spare or thin, belonging to what some call the nervous type. The patient is stated to be thin in 59, and the nutrition moderate in 48, whilst the patient was *stout* in only 16 instances. As regards vigour, the conditions are much the same; the patient was weak in 40 cases, moderate in 57, and strong only in 24.

The *complexion* was fair in 68, dark in 52, ruddy in 1, and in 1 mixed.

The *previous mental condition* was good in the vast majority of cases: good or normal in 100, sharp or quick in 4, precocious in 2, moderate in 1, sluggish in 2, weak in 6.

The *growth* was moderate in 50, rapid in 55, slow in 17.

*Menstruation*.—Many of the females had not reached the menstruating age. Of the recorded cases it was regular in 13, irregular in 10, one was pregnant, and in one it had ceased.

The next point is one of much interest and importance, viz., *antecedent illnesses*.

Definite *rheumatism* preceded the chorea in 31 cases, and in one more there is a query to the reply. This would give a percentage of definite antecedent rheumatism of 24·22. Of the 31, it is stated that there had been "rheumatic fever" or "rheumatism with fever" in 16, "rheumatism with joint affection" in 7, and simply "rheumatism" in 8. If to these 31, three cases in which rheumatism is given as the exciting cause, in cases in which there was no rheumatism antecedent to the exciting cause, and 4 cases in which rheumatism occurred during or after attacks of chorea without having been antecedent, it would bring the number up to 38, or 30 per cent. (29·61) in which chorea and rheumatism were associated. "Vague pains," or vague rheumatic pains are recorded in 15 cases, exclusive of those in which definite rheumatism had occurred. The significance of these vague pains is known, much debated, and all the cases in which they occur will be submitted to most rigid scrutiny by the Committee in the final report, so that any bias of those holding opinions on the point may, if possible, be avoided. It may be mentioned, however, as important, that, in 8 of the 31 cases in which definite rheumatism is said to have anteceded the chorea similar vague pains occurred. Scarlet fever occurred as an antecedent illness in 41, measles in 17, whooping-cough in 5, enteric fever in 5, tonsillitis in 3, abscess in ear in 1, congestion of brain in 1, neuralgia in 1, eczema in 1, pleurisy in 1, worms in 1. Anæmia had been present in 28 cases. The period it had lasted is stated in most cases, and its degree in many. It appears to be a powerful predisponent. A point likely to be determined in the final report is the *rôle* played by anæmia in the production of heart-disease. It may be mentioned meanwhile that there was heart-disease in 7 cases of anæmia in which no rheumatism is recorded.

*Exciting Cause.*—An exciting cause for the attack is mentioned in 104 of the cases. As might be anticipated, fright predominates under this head, being mentioned in 29 cases. In some of these the nature of the fright is clearly defined, and its influence able to be gauged. In many, however, it is simply stated “fright,” which is too indefinite to be wholly trustworthy. It is hoped that the future cards will give the information clearly but briefly. Overwork figures next as occurring in 23: these are mostly overworked school children, and the matter is of grave importance. Bodily overwork occurred in 10 cases. Shock is given as the exciting cause in 6, anxiety in 3, grief in 1, and the attack is ascribed to “love”—it must be presumed the love was disappointed or unreciprocated—in 1 case. Imitation is blamed in 4 cases, and worms in 4. In 2 cases chorea followed falls after intervals of 3 days and 2 weeks. One of these cases was a girl of 18, in whom the attack came on 2 weeks after a fall; she had not had rheumatism, but had scarlet fever in infancy; the heart was normal (Mr. R. W. Barrow, Liverpool). The second was a girl of 9, who fell 10 feet from a ladder, striking her forehead. Epistaxis followed, and chorea, severe in degree and lasting 3 months, followed the fall by 3 days. She had not had rheumatism, and there was no heart-disease except palpitation (Dr. A. R. Mackenzie, Fortrose). In another case in which the attack was induced by the fright from a large dog, a previous attack had followed a fall (Mr. J. Ward, Merthyr). Heat, sunstroke, enteric fever, debility, and dysmenorrhœa, are returned as exciting causes in 1 each, and cold and dyspepsia 2 each. In many of the cases more than 1 cause was, or appeared to be, contributory to the incitement of the disease. The disease occurred in the course of rheumatism in 11 cases, two of them being post-scarlatinal. In some of the cases the disease is stated to have changed from rheumatism to chorea; in others intervals of from a few days to some weeks occurred between the rheumatism and chorea. In one case fifteen months is stated to have elapsed between the rheumatism and chorea, though the former is returned as the exciting cause. This is probably an inadvertence: the rheumatism may have been a *predisposing* cause, but it would be inexact to record it as the exciting cause. In one case under exciting cause is given “a scolding” rheumatism. As in this case the patient



had previously suffered from rheumatism, and during the attack of chorea had an attack of acute rheumatism affecting many joints, had heart-disease previous to the attack, and subcutaneous nodules during it, it is clear that rheumatism was an important element in the case. But as it is complicated by the "scolding," it is not included in the cases of rheumatism exciting the attack. Greater accuracy is needed in filling up the reply to exciting cause: in some cases the cause stated is too vague to have any meaning, and in others the interval between the supposed cause and the attack is so long as to remove the cause from the category of exciting. It is, of course, of great importance to ascertain whether the patient has had *previous attacks* of chorea, and much useful information has been elicited as to former attacks in the same subject. The majority of the cases were first attacks, but there were 12 second attacks, 2 third attacks, and 1 fourth attack. The returns as to the attack being febrile or non-febrile have been moderately complete, but some have not answered this question. So far, the non-febrile preponderate considerably over the febrile.

With reference to the occurrence of *heart disease* in connection with chorea, the general tendency of the returns will be shown by the statement that in 33 cases, or 27·34 per cent., heart disease was found to be present. This is excluding all functional derangements and doubtful cases. In 10 of the cases the heart was known to be affected previously; in 15 it was known to be normal at some (unrecorded) prior date, and in the remainder its anterior condition was unknown. The heart remained affected in 26 cases. The lesion in nearly all cases was mitral regurgitation; in one case there was pericarditis.

Rheumatism occurred during the attack in 23 cases. In 19 of these cases the patient had suffered from rheumatism previously, and in two more antecedent vague pains are recorded. Other complications mentioned are—urticaria in 2, constipation in 2, delirium, epileptic fit, aphasia with right hemiplegia, headache, facial neuralgia, syncope, peritonitis, apical pneumonia, each once. Subcutaneous nodules are recorded in three instances. (Dr. Bevan, Mumbles; Dr. Harkin, Belfast; Mr. Messiter, Dudley.) In all of the cases there was coincident rheumatism, and all had suffered from previous rheumatism. A point of much practical importance, however, is that in two of the cases there was no

affection of the heart. Only one death is recorded, and this in the quite exceptional cases alluded to later in a patient 78 years of age.

*Common ailments.*—It was hoped that valuable information on this point would be elicited in order that the clinical relationships of chorea might be ascertained. These returns are not so complete as could be desired, but they do afford instruction. It is elicited that headache is frequent in the choreic, being recorded in 21 cases. In some it is described as “migraine,” in others as “sick headache,” “bilious headache,” “repeated severe headache,” &c. Neuralgia occurred in 4 cases, urticaria in 3, incontinence of urine, masturbation, stammering, asthma, each in 1, besides nervousness and hysteria—all these being evidences of chronic or occasional nervous derangement. Dyspepsia is recorded in 5, tonsillitis in 3, catarrh in 3, bronchitis in 2, and cough in 1, besides numerous other single ailments. The only skin disease mentioned besides urticaria is eczema in 1 case; no cases of erythema nodosum, erythema papulatum, or other forms of erythema are recorded. One patient had an attack of pityriasis rubra whilst in hospital, and the temperature ran up to 104° F. (Dr. F. Pollard, Liverpool).

With reference to *nervousness and nervous affections in the families of those suffering from chorea*, the returns supply information which strengthens the general opinion that the disease is especially apt to occur in families in which other nervous disorders are common. In 78 members of the chorea patients' families nervous disorders or affections are recorded. In one case, the patient's age being 43, his son had chorea; the father had suffered from rheumatic fever, but the son had not (Mr. Gowans, South Shields). In the families of the choreic, rheumatism, as might be expected, is pretty common, as is borne out by the replies. Gout, another neuro-humoral disease, also figures in some of the chorea families.

The returns on sequelæ do not contain much information of importance, though some exceptional cases of great clinical interest are recorded—notably one by Dr. Walter Bernard, of Londonderry, who sends a cast, taken by himself, of a hand deformed by chorea. This cast exhibits typically the “main griffe” of “progressive muscular atrophy;” it was no doubt

produced by the choreic paralysis and subsequent atrophy of the interossei muscles; as this paralysis tends to recover, it was anticipated that the hand would improve, and this prognosis has been borne out by the subsequent history of the case.

The records of *treatment* contain much of interest and importance. Sulphate of zinc appears to have been of striking utility in some cases, and chloral hydrate in others. Arsenic appears to have been given in the majority of cases with varying degrees of success.

The cases occurring at exceptional ages, already alluded to, are (reported by Dr. F. Aitken, Inverness) in a patient of 86, in which the disease is stated to have been severe and to have lasted for about one year; (reported by Mr. Bridger, Cottenham) a case of a woman, aged 78, in which the choreic movements were chiefly left-sided, and in which ulceration of the tongue occurred in consequence of the choreic movements; the disease did not last for more than a couple of months; the patient suffered from chronic rheumatism; the heart's action was weak, but there was no bruit—the case terminated fatally. The third case (reported by Mr. Gowans, South Shields) was in a man 43 years of age, who is stated to have suffered from rheumatic fever and vague pains, and to have had heart dropsy; he had tricuspid disease and hypertrophied right heart. It is the case, previously mentioned, in which the patient's son also had had chorea. The Committee will endeavour to obtain further information regarding these unusual cases, and in other cases presenting peculiar features.

It must not be concluded from these observations that any question on which information was needed, or concerning which divergences of opinion exist, is set at rest by this brief and incomplete analysis of some of the returns. Enough has been said, however, it is hoped, to show that the returns are of real value, that the subject is one well adapted for collective investigation, and that, if there is a large augmentation of the number of cards, fully and carefully answered, the investigation will prove a solid contribution to clinical medicine.

To recapitulate the points on which further, or more accurate, information is desired:

The sex and age to be stated in all cases. All the cases in reporters' practice should be recorded, if possible, so that accurate



information may be gained as to class incidence. Information as to prevalence of chorea in district of reporter is of the greatest importance, and will be highly appreciated by the Committee.

As regards exciting causes, greater exactness as to exciting causes (when due to fright its exact nature to be stated), and as to the interval between the exciting cause and the onset of chorea.

More complete information as to the condition of the heart. It is stated on several of the cards that the heart was too tumultuous to be examined during the attack; but it can be examined and reported on *in every case* at the termination of the illness.

More information is greatly desired as to the common ailments to which chorea patients are subject.

*The Sub-Committee is indebted to DR. STEPHEN MACKENZIE  
for the preparation of this Report.*

(Signed) THOMAS BARLOW, M.D.  
DYCE DUCKWORTH, M.D.  
J. F. GOODHART, M.D.  
STEPHEN MACKENZIE, M.D.  
F. A. MAHOMED, M.B., *Secretary  
to the Committee.*

## A PRELIMINARY REPORT ON ACUTE RHEUMATISM.

It is not proposed on the present occasion to deal at any length with the material received concerning acute rheumatism; this will form the subject of a subsequent report. It may meanwhile prove of some interest if a short account is given of the returns received, indicating in what directions they are likely to advance our knowledge of the disease, and also suggesting some points for future observations.

Up to the end of June, 1883, 339 returns have been received. Of these 208 only are dealt with on the present occasion, which happen to have been tabulated in a form easy for reference.

It is impossible to study these returns carefully without being convinced that we are dealing with a mass of information of very great and exceptional value. There is perhaps nothing in the history of medicine strictly comparable with it. These returns are not the observations of any single person, who may have been more or less biassed in the selection of his facts or his cases; they are not mere chance reports which have been made with no particular object, and which are subsequently referred to in the hope that certain points may happen to be mentioned in them; nor are they observations made on one or more points by a single individual who may have been anxious to prove some preconceived theory or to support some other antecedent observations. They consist, on the other hand, of cases recorded by a large number of individuals who have previously been asked to make particularly careful observations with regard to certain features of the disease. Moreover, they are not cases which have merely passed under observation for a short while in hospital, but they mostly consist of records of the incidence and progress of the disease in persons whose antecedent

and subsequent history is well-known to the observer; they therefore afford information that cannot be obtained with regard to hospital cases. It should also be remembered that these returns are contributed by a selected body, and the selection has been a "natural" one.

We may now proceed to consider our material in more detail. It affords a history of the disease, replete with exact statistical details, which far surpasses in the amount of information which it contains any account of acute rheumatism with which we are acquainted. The returns will give replies to various questions with statistics, which will more or less finally settle these questions; whether this result is achieved or not depends on the numbers we may yet receive, which we hope will bring the aggregate returns up to 1000.

We may first seek for information concerning the incidence of the disease upon the sexes, and the ages between which it is most frequent in each sex. Out of the 208 cases tabulated, in one case the sex is not stated, and the age is not stated in the cases of three males and one female; and we have therefore only 203 cases to deal with, and they give the following result:—

*Table shewing the incidence of Acute Rheumatism as to age and sex.*

Age.	Male.	Female.	Total.
5 years and under . . . . .	1	1	2
6—10 . . . . .	7	2	9
11—15 . . . . .	6	18	24
16—20 . . . . .	16	17	33
21—25 . . . . .	27	17	44
26—30 . . . . .	18	10	28
31—35 . . . . .	16	7	23
36—40 . . . . .	9	6	15
41—45 . . . . .	7	3	10
46—50 . . . . .	1	4	5
51—55 . . . . .	3	2	5
56—60 . . . . .	1	1	2
Over 60 . . . . .	2	1	3
	<hr/> 114	<hr/> 89	<hr/> 203

These figures, though small, are probably fairly correct; they agree in the main with former statistics and common experience, but they bring out one or two points of interest. They show that,



taking the two sexes together, a larger number of cases of acute rheumatism occur between the five years from 21—25 than at any other period; the numbers rising rapidly from 10 up to 25, and decreasing more slowly from 25 to 45; the ten years of greatest frequency of the disease is from 16—25, during which 77 cases, as compared with 51 cases from 26—35. The disease is obscure or rare under 5, whilst it is infrequent after 45 years of age. When we compare the incidence of the disease upon the sexes, some points worthy of note are observed. Between the ages of 11 and 15 it is three times more frequent among females than males; between 16—20 the numbers are practically equal; while after this age the disease is far more frequent among males than females, up to the age of 45, when they again become equal, 7 cases occurring among men and 8 among women, while between the ages of 20—45, 77 cases occur among men and only 43 among women. Or, the difference in the sexes may be expressed in another way,—women become susceptible to acute rheumatism five years earlier than men, but men continue susceptible for ten years longer than women; males have, moreover, a period of special susceptibility between the ages of 21—25, while there is no corresponding short period of greatest susceptibility among women. It may be that these figures are too small to draw any conclusion from; if, however, these indications are true, it would appear that possibly the onset of menstruation may increase the liability to the disease, while child-bearing would seem to protect from it, or else that the increase of the disease in men after 20 is due to their increased liability to exposure. These questions may perhaps be decided by information afforded under other heads, among others, occupations—which is the next question on the card—may become important.

The returns concerning those who are described as intemperate will have to be examined very critically with the view of ascertaining whether the disease is more fatal, more prolonged, more complicated, or the recovery less complete with them. It is certain that total abstinence does not protect from it.

The next question, that as to residence, is one of great importance, and with regard to this, as also in the case of chorea, it may prove necessary to issue some special inquiry. We should

like to ask every practitioner in the Association to note the number of cases of acute rheumatism (and also those of chorea) that fall under observation in the course of one year. This would place at our disposal most valuable data, from which it might be possible to determine the geographical distribution of the disease, and its connection, if any, with variations of soil and climate; it is scarcely possible to do this satisfactorily from the Registrar-General's returns, on account of the very low mortality of both acute rheumatism and chorea. There is another plan which we have in contemplation, as perhaps more easy, though not as accurate, namely, to apply for returns from all the hospitals, cottage hospitals, dispensaries, and infirmaries throughout the British Isles, asking for the number of cases of each of these diseases admitted during one or more given years.

With regard to the next question, as to locality of residence, whether exposed, shut in, on high or low ground, &c., the returns may yield some information which, even if it be of a negative character, will be valuable. So also as to the prevailing wind and atmospheric conditions at time of onset. These may show how much "exposure" may act as an exciting cause of the disease; we know that this cannot be the ultimate cause, though it may often be the proximate one. That acute rheumatism is a diathetic or constitutional disease is proved by its frequent occurrence in one person, while others escape altogether, by its hereditary character, and by the diseases that are associated with it.

It would seem that this constitutional condition can be readily induced in a person not previously disposed towards the disease; the manufacture of rheumatism is seen in a striking manner during scarlatina, of which we shall probably have more to say hereafter. The Committee would take this opportunity of asking for careful records of any cases of acute rheumatism occurring after scarlatina; it is known that these cases are commonly so slight as to be better described as subacute than acute; and this is probably the reason that has almost excluded them from the returns. But however slight they may be, if well-marked and unmistakable, they would be gladly welcomed; the Committee would especially appeal to the physicians and resident medical officers of fever hospitals to help them, by recording some of the many cases of the kind which they see. Much of scarlatinal

rheumatism, in our own experience, is only muscular, but it is nevertheless susceptible to immediate relief by the salicylates; even these cases might be recorded, though they may have to be dealt with separately in the final report.

Passing now to the replies received to the question concerning "recent antecedents," one point appears to come out strongly, namely, the frequency with which tonsillitis precedes acute rheumatism; this is a relationship which has been already remarked by several writers, and deserves much further consideration. In no less than 57 cases out of 208 cases, that is, in over 25 per cent, tonsillitis (or, in one or two cases, pharyngitis) is stated to have preceded this disease. It must, however, be mentioned that the periods antecedent to the rheumatism at which the tonsillitis occurred are variable; these will demand further consideration.

"Exposure" and "over-fatigue" are reported to be very frequent antecedents, thus giving support to the generally accepted doctrine. Exposure to *wet and cold* is given as an immediate antecedent in no less than 85 cases, that is, in nearly 41 per cent. In this number a very few are described as exposed to "cold" only; they are nearly all "cold and wet"; a few "wet" only. Those in which the exposure is described as "constant" and "very frequent" have been excluded. The precise dates of exposure are given in nearly all the cases; and in the final report these details will be worked out. But another element enters into these exciting causes, namely, *fatigue*. In 30 other cases out of the 208, that is, in 14·4 per cent., wet, cold, and prolonged or severe over-fatigue are all given as immediate antecedents; while in 19 others, or in 9·1 per cent., "over-fatigue" alone is registered as an exciting cause. Thus, in 23·5 per cent. of the cases "over-fatigue" plays a prominent part; and the two conditions of "exposure" and "over-fatigue" immediately precede no less than 64·5 per cent. of the cases. Some of these also suffer from "tonsillitis," which in many cases follows the exposure.

Some interesting statistics of the clinical history of the disease itself, as to the duration of fever, duration of pain, and of the whole illness will be forthcoming; and a comparison of various



methods of treatment will be possible. This has not been attempted with our present material; it will be an investigation of much labour, and must be done thoroughly when taken in hand. Unfortunately, the replies in this direction will not be as perfect as could be wished, chiefly because the questions with regard to treatment are not so precise as they should have been; consequently, the observers, while stating in all cases the general line of treatment, have generally omitted to state how long the medicine was given, many have not stated how frequently it was taken, or even in what doses. It will very greatly facilitate the work of the Sub-Committee if, in cards yet to be filled up, the observers are careful to *state* (1) *in what doses the drugs are given*, (2) *how frequently administered*, and (3) *over what periods they were continued*. The favourite drug appears to have been salicylate of soda; but a sufficiently large proportion of cases have been treated by other methods to enable a fair comparison to be drawn between the results obtained. No less than 116 out of the 208 cases already tabulated were first attacks, and these will furnish some very important evidence as to the degree in which treatment by salicylates prevents heart complications, especially as it may be possible by additional inquiries to ascertain in many cases what is the condition of the heart at some considerable period after the disease.

There are many exceedingly important and interesting points to be worked out with regard to the relation of heart disease to rheumatism. Evidence has of late been accumulating which shows that the generally accepted doctrines on this subject by no means cover the whole ground. Many other things besides the so-called rheumatic inflammation of the valves may bring about valvulitis and subsequent incompetency. It would seem that dilatation of the ventricle is in many cases the first step; this may be due to mere muscular debility, to anæmia, with or without fatty changes, besides impairment of the muscle of the heart by pericarditis and myocarditis, and strain thrown upon the ventricle by increased arterial pressure. When the ventricle dilates, either temporarily or permanently; greater strain is thrown upon the valves, which are required to close a larger auriculo-ventricular opening than normal; smaller surfaces of their corre-

sponding cusps are brought into contact; their *columnæ carneæ*, which tend to diminish the strain upon them, especially by counteracting the tendency to eversion, act less powerfully and effectively; consequently much greater strain is thrown upon the valves, and chronic valvulitis is set up. If these abnormal conditions are maintained, the disease advances, and the valve rapidly becomes incompetent. This may be the cause of heart disease in many rheumatic subjects, who are, no doubt, particularly liable to valvulitis, though this probably has no specific character; this theory of increased strain upon the valves in weakly and anæmic persons accounts for many cases in which the heart is found to be diseased before the patients have had any attack of acute rheumatism; heart disease is stated to have been present before their illness commenced in *four* of the patients whose first attacks of rheumatism are recorded in the returns. Opportunities not infrequently present themselves of watching the evolution of heart disease in cases in which displacement of the apex beat is the earliest symptom, and occurs before any valvular murmur; this is especially frequent in anæmia, diphtheria, scarlatina, and Bright's disease, and in choreic subjects.

Of course, in such cases as these, the treatment of acute rheumatism by the salicylates could have little or no influence on the development of the heart disease which is produced by the condition leading up to rheumatism, or is the result of the anæmia and debility preceding or following that disease.

One of the most important questions on the card is that which refers to "minor ailments" to which the patient is commonly liable. It is to these minor ailments that we are most anxious to call the attention of the profession. They have never been sufficiently studied; there can be little doubt that they mark the diathesis or constitutional predisposition of individuals, but as yet we have but little evidence, though many general impressions. It is our belief that most people have some form of common ailment, but it is remarkable that a very large number in these returns are said to have none; we fear that this may mean that they were not sufficiently inquired for. Those that are reported are strongly in favour of the neurotic relationships of rheumatism; out of 208 cases we find sick headaches recorded in 14, neuralgia

in 12, urticaria in 3, goître in 1 (in a girl of 12), making in all 30 cases of a neurotic type, or 14·4 per cent. In 18 other cases, tonsillitis is recorded as the prevailing minor ailment.

Of the skin diseases recorded there is not much to say at present; urticaria, as might have been expected, heads the list with 7 cases, some of them occurring during the attack, 4 cases of eczema, 2 of erythema, 2 of purpura, and 1 of psoriasis are mentioned. In 3 cases subcutaneous nodules have been observed.

A point of considerable interest comes out in the large number of cases in which disease is said to have persisted in one or more joints. This is reported in no less than 33 cases. With regard to these it will be necessary to make further enquiries of the observers, as to how long the joint affection continued to persist, and whether in any cases it advanced to disorganisation, or to any surgical affection of the joint. It is believed that surgeons are more familiar with joint disease resulting from rheumatism than physicians expect; it would be very valuable if some more correct opinions could be formulated as to the probability of such a result, and definite lines be laid down for its treatment in the early stages.

These 208 returns contain 8 fatal cases, giving a mortality of 3·84 per cent., probably a rather high one, as there is a tendency to record interesting cases rather than those of the ordinary type. The causes of death in these cases are important, and we may have to ask for further details before publishing the cases in full, as we shall hope to do hereafter. In 3 of the cases, delirium was a marked symptom, without hyperpyrexia, and our own experience strongly confirms the gravity of this symptom in acute rheumatism. In another fatal case dilatation of the heart is stated to have produced death, and this in a first attack. Two very remarkable cases of hyperpyrexia are recorded by Dr. Homan of Lichfield, in which both father and son died from hyperpyrexia in acute rheumatism within a few days of each other; these, with other cases of great interest, will require further detail and comment in our Report. No more has been attempted on this occasion than to indicate the value of the returns received, and the sort of information that is obtainable from them. This has been done in the briefest possible manner; many other points of interest will become apparent by a more



careful and elaborate comparison of the returns; we trust that sufficient has been said to encourage those who have helped in this work to continue to give their assistance, and to give evidence to others who have not yet contributed, that any trouble they may take will not be thrown away. It is hoped that when the final Report is presented a tabulated record of all the returns received will be given.

*The] Sub-Committee is indebted to DR. MAHOMED for the preparation of this Report.*

(Signed) THOMAS BARLOW, M.D.  
DYCE DUCKWORTH, M.D.  
J. F. GOODHART, M.D.  
STEPHEN MACKENZIE, M.D.  
F. A. MAHOMED, M.B., *Secretary*  
*to the Committee.*

## A PRELIMINARY REPORT ON DIPHTHERIA.

THE Committee on diphtheria are glad to be able to report that notwithstanding the difficulties which surround the investigation of their subject, an amount of valuable information has already been received, leading them to hope that a continuance of the labours of the investigators will eventually throw light upon some points in the behaviour of this disease. At this moment, however, they are unable to do more than indicate the directions in which the communications already received appear to lead, leaving to a later date a more precise statement.

Altogether, 138 communications have been received, 64 of which give information concerning the sanitary and clinical features of each case, 26 giving information on sanitary points alone, and 48 on clinical points alone; several of the communications relate to groups of cases.

It is interesting to note that three-fourths of the cases which have been reported are described as *sporadic*, originating without known exposure to any antecedent case, and that by far the greater number, whether sporadic or epidemic, have occurred in sparsely inhabited districts, a point particularly noticeable among the former class.

The opinion has long been held that diphtheria is a disease especially of rural districts, but the evidence with regard to the relation of its prevalence to population has until now never been satisfactorily shown; the Committee are, therefore, glad to be able to refer to a paper which has been prepared for them by Dr. G. B. Longstaff, which, dealing with the matter as one of figures alone, proves conclusively that the greatest mortality from diphtheria occurs in thinly inhabited areas. It is intended hereafter to publish Dr. Longstaff's valuable contribution; it will now suffice to state that he found that while 62 per cent. of the

total districts in England and Wales had a mortality from diphtheria above the average in the decades 1851—60, or 1861—70, or in both periods, of the densely populated districts only 36 per cent. were above the average.

When allowance is made for the fact that in thickly populated areas diphtheria must have, through personal communication, especial opportunities for extension, it is somewhat striking to find that some condition peculiar to thinly populated areas should obscure the effects of this opportunity.

With regard to soil it is interesting to note that two-thirds of the cases have been situated upon the clay, and that this rule has been followed by sporadic cases only. In one half the country has been described as hilly, while of the remaining three-sixths, in two, the country has been undulating, and in one, flat. In the greater number of cases the infected house has been situated on the hill-top and on the hill-side rather than in the valley, and wherever placed, the soil was more frequently wet than dry.

Concerning seasonal prevalence, about four times the number of sporadic cases have occurred in the winter months, October to March, as compared with the summer months, April to September. This agrees generally with a diagram published in the Registrar-General's Annual Summary for 1880, showing the curve described by the weekly mortality from diphtheria and based on the records of the twenty years 1860-79. It would be interesting to note whether there is any difference between the seasonal prevalence of cases resulting from communication of infection from person to person and those which apparently arise without such cause. If so, there is a greater probability that some local cause other than an antecedent case may give origin to diphtheria.

In connection with the winter prevalence of diphtheria it may be stated that fully three-quarters of the cases communicated occurred at a time preceded by a condition of the atmosphere which is described as "damp," two-thirds of the cases having in the preceding month been attended by "excessive" rainfall.

Of the cases in which this information is given, in rather more than one-quarter was scarlet fever said to be recently prevalent in the neighbourhood. In about the same proportion of cases, the same fact was observed with regard to diphtheria, while in but four cases were both diseases stated to have prevailed. It will



be obvious that each investigator would probably not always be able to speak with any certainty as to the prevalence of these diseases in other houses than the one he was visiting, and too much importance must not, therefore, be attached to these replies, and so far no very positive information is forthcoming in favour or against the belief held by some that there is a special relation between scarlet fever and diphtheria. Evidence which Dr. Longstaff brings to bear on this point has, however, an especial value, for a continuance of his inquiry already referred to shows that the relation of diphtheria to density of population is almost exactly the converse of that of scarlet fever. He submits the following hypotheses as both explanatory of this fact. Firstly, scarlet fever and diphtheria are entirely distinct diseases, though having many points of resemblance; in the case of scarlet fever, the disease being mostly spread by personal intercourse; in diphtheria, the disease mostly spreading in some indirect manner, possibly frequently originating *de novo*. Secondly, scarlet fever and diphtheria are two forms of the same disease, the first being met with more frequently in crowded towns, whereas the same poison in country districts more frequently produces the second form.

Important evidence against the second hypothesis is, however, afforded by the communications already received, which show that where definite information is given as to the illnesses from which those attacked with diphtheria had previously suffered, one-half of the cases had previously passed through an attack of scarlet fever. Now it cannot be denied that some persons suffer from more than one attack of the same infectious disease, but it is in the highest degree improbable that these persons should number 50 per cent. of the community. With the question in this position it is deserving of notice that in but two cases did a definite exposure to scarlet fever precede the attack of diphtheria; in one of these cases several members of one family were attacked with illness, in all but one the disease was called scarlet fever, in the remaining one diphtheria. When due allowance is made for the difficulties of diagnosis in some cases between scarlet fever and diphtheria, it is clear that information on this point to be of value should show whether the subsequent appearance of albuminuria during convalescence, or the free desquamation of the skin, leads to the opinion that a case occurring under these circum-

stances is scarlet fever, or whether a subsequent paralysis indicates that the patient suffered from true diphtheria.

Concerning croup, definite contact with this disease is stated to have occurred in two cases, with "croup and diphtheria" also in two cases, while "croup" in one case, and "croup and scarlet fever" in another case, were said to prevail in the neighbourhood at the time of or just before the attack.

The proportion of cases who had blood relations who had previously suffered from diphtheria is small, not exceeding one-fifth; in about one-fourth, sore throat had frequently existed in the house where diphtheria subsequently occurred, and in one-sixth other persons in the house suffered from sore throat just before or during the illness chronicled. This antecedent sore throat, that was not recognised as diphtheritic, is of great interest and importance. It might seem to point to the gradual evolution of the specific disease. It is most probable that the Committee will desire to make further inquiries in this direction.

As to methods of dealing with excrement, the Committee have as yet no evidence before them which would sufficiently show whether there is any special incidence of disease upon houses or districts where there is one or another mode of treating excreta.

Again, with regard to water supply, no case has as yet been brought to the notice of the Committee in which a number of persons have been simultaneously attacked with disease in the manner that experience of other affections, such as enteric fever, shows to be usual when the illness arises from a contaminated water-supply used by a number of persons.

Only 38 replies have been made to the question whether any disease of the lower animals had prevailed in the neighbourhood where diphtheria had occurred; in 29 it was stated that no such disease had been present; in five there had been foot-and-mouth disease; in one, fluke-rot among the sheep; in one, scab among the sheep; in one, disease among the fowls; and in one, it was stated that a horse in adjoining stables had suffered from an affection of the throat described by the veterinary surgeon as diphtheria.

With regard to the local conditions which are by some believed to cause diphtheria, it is important to note that in less than one-tenth was the disease attributed to a defective drain or sewer. It

must not, however, be understood that what is technically known as a nuisance was not found in connection with a large number of houses in which disease occurred; but even when faulty dust-bins, offensive yards and piggeries, offensive drains, or mould upon a wall, are taken into consideration, more than half the houses were absolutely free from any such defect, while of those which were open to this accusation but one-fifth were found to have mould on the walls. It is probable that at least the same conditions would be found in not smaller proportions in houses altogether free from disease, and there does not therefore as yet appear that any such local condition can be regarded as a cause of diphtheria.

The material in the possession of the Committee, as a rule, only enables the longest possible incubation period to be noted, the duration of exposure to infection having in almost every case lasted some time. It may, however, be stated with this limitation, that the periods have extended from a few hours to twenty-two days. Further information on this point is much needed, and should always include the longest and shortest possible period of incubation in each case, where the exposure to infection has been of a continuous character.

The chief clinical features of the cases communicated may be briefly noted:—glandular swelling was stated to be present in 100 cases, and in no case was its absence recorded.

The presence of a membrane was almost equally constant; in but one case was it stated to have been absent; in half the cases it appeared on the first day of illness; in 68 per cent. it was not delayed beyond the second day, and in 86 per cent. not beyond the third, while the latest day of its first appearance was the fifteenth day of the illness. Its duration lasted varying periods, from one to twenty-six days, the average duration being about eight days.

Of the total number of cases of diphtheria paralysis is said to have been present in twenty-five, but concerning many no statement is made. In 19 the muscles of the soft palate or of deglutition were affected, in one there was paralysis of the larynx, in another of the diaphragm, in three there was loss of sensation and taste, and in one the site of paralysis was not mentioned. The duration of this symptom lasted during periods extending from three days to five months.



Albuminuria was present in 88 per cent. of the cases in which its presence or absence was recorded; these, however, constituted but a third of the total cases. This symptom appeared as early as the first day, and as late as the sixteenth, and in 85 per cent. within the first week; its duration was from two to sixty-three days, in 71 per cent. being under a fortnight.

Some very interesting details of an epidemic which occurred at Magdalen, near Downham Market, have been communicated to the Committee by Dr. L. King; this epidemic was remarkable for its clinical features; the presence of membrane being by no means constant, while the kidney complications were remarkably severe.

Other valuable reports concerning epidemics have been received from Dr. Fussell, Medical Officer of Health for East Sussex, from Dr. Barnes of Eye, Dr. Tylecote of Sandon, Dr. H. S. Renshaw of Manchester, and others. These will be dealt with hereafter, but the Committee would take this opportunity of asking Medical Officers of Health and others to furnish them with any points of interest that come before them in connection with this disease.

The number of cases does not warrant any conclusions being drawn as to the advantages of any special mode of treatment; a further discussion of this branch of the subject must, therefore, be deferred for the present.

The Committee, in conclusion, desire to express their obligations to the different investigators for their communications, and trust, as a result of further information, to be able, in a subsequent report, to consider more in detail the various points of interest.

*The Sub-Committee is indebted to MR. SHIRLEY MURPHY  
for the preparation of this Report.*

(Signed) F. P. ATKINSON, M.D.  
THOMAS BARLOW, M.D.  
ALFRED CARPENTER, M.D.  
W. B. CHEADLE, M.D.  
SHIRLEY F. MURPHY.  
A. RANSOME, M.D.  
F. A. MAHOMED, M.B., *Secretary  
to the Committee.*

## Subject for the Next Inquiry.

---

### THE TREATMENT OF ACUTE GOUT.

#### MEMORANDUM AND CARD.

AMONGST other inquiries which the Collective Investigation Committee have determined to make at the hands of members of the Association is one having reference to the treatment of Acute Gout.

For this purpose a short therapeutical card is now issued.

The subject is one of large interest to all British practitioners, and although it may be thought that there is no pressing need for information upon the treatment of acute gout, we hope to show that this is not the case, and that, on the contrary, there is still much to be learned, and much of practical importance which may, without great effort, be ascertained.

The inquiry is issued with especial reference to treatment, and it is limited to cases which present no difficulty or ambiguity in diagnosis. Where a practitioner is called to a case of classical acute gout, affecting the ball of one big toe, he can forthwith begin to fill up the card.

It is certain that such cases are not met with frequently in hospital practice. Acute gout may supervene in either a medical or a surgical ward; but it is then seen as a complication of some other condition, and can hardly be studied to full advantage for the purpose of an inquiry such as this. Cases are usually not seen amongst out-patients till several days of the acute attack have passed, when the pyrexia has subsided, and, often, most of the pain. Much can be learned from such cases, but certainly nothing in respect of the true value of rapidly applied and skilfully administered therapeutic effort.

The natural history of acute podagra, unsubmitted to treatment, is well-known. What is not sufficiently known is the line of treatment to pursue. It is not to be supposed that the Committee desire to set the members of the Association in search of a specific for all cases, though Heberden,\* more than a century ago, said that he hoped the time would come when the discovery of a specific for the gout, as certain as those which had been found for ague and itch, would shew the equal safety and advantage of immediately stopping its career and preventing its return. Some are of opinion that we already have a remedy of marked specific power in colchicum. Others can tell how they rob gout of its terrors by hypodermic injection of morphia, which certainly is not without startling effect in some instances, and which, as Mr. Teale suggests, indicates a line of thought respecting the neurotic theory of the malady. Others, again, profess to have discarded colchicum since they became possessed of a talisman in the shape of the salicylates.

This inquiry will, without doubt, elicit replies indicating faith in many modes of treatment; and the Sub-Committee of Inquiry will find, very likely, that to many minds the question is solved by methods so various as Turkish baths, firm local strapping with diachylon plaster, bromide salts, and lithium combinations. The public in its agonies has gone far afield for solace, as is well known, and has sought it in teetotalism, vegetarianism, dietetic experiments of all kinds, and lastly in hot water.

This diversity of opinion and treatment cannot be considered satisfactory or becoming to the scientific spirit that is supposed to pervade the practice of physic at the end of the nineteenth century.

In the card there are a few points of clinical inquiry which are necessary to make the reports of value. It will be well to direct attention to some of these. First, in respect of the patient's residence. It is a common belief that there is more gout, that is, unequivocal gout, in the south than in the midland counties and in the north of England; also, that in Scotland and Ireland the disorder is practically unknown amongst the middle and lower orders.

\* *De Arthritide Commentarii.*



To connect residence with occupation will prove of further interest. The influence of close confinement, sedentary occupation, and, in particular, the effect of exposure to lead-contamination, will be elicited. Respecting the latter, it is truly remarkable that the facts illustrating the intimate connection between lead-taint and gout are derived almost solely from London practice, the experience of the great manufacturing centres elsewhere in this country and on the continents of Europe and America giving no support to it.

Dietetic habits necessarily command marked attention. Knowledge is wanted as to the particular alcoholic beverages which seem most to provoke gout. The last word has certainly not yet been said upon this subject. It is not too much to aver that the variance of opinion in the profession as to the qualities and properties of alcoholic drinks generally, and as to their fitness for treatment of many morbid states, is eminently unsatisfactory and unworthy of men whose life consists mainly in observation. Attention is specially directed to malt liquors and to that atrocious compound, so largely imbibed by the labouring classes, "four ale." This liquid is known to be impregnated with lead in small quantity.

The immunity from gout enjoyed in Germany, where beer is more largely consumed than here, and the absence of the malady from the Russian lower orders—the most drunken in Europe—indicate that much has yet to be learned before it can be affirmed with confidence that gout is due to alcoholic intemperance.

It is sought to discover further, how much fluid of all kinds is commonly taken daily by the gouty. It is asserted that many of these patients rarely take any but strongly impregnated liquids, and but seldom drink water.

The influence of meat-eating is likewise of importance to note. Thus, it is certain that more butcher's meat is consumed in England than in the other divisions of the kingdom.

Attention is directed to the habits of exercise, and this will bear in part upon another point, viz., the action of the skin. In some persons there is very small sudoriferous waste. Many gouty persons have inactive skins in all but the warmest weather, when they perspire profusely.

It is very desirable to gather knowledge as to the type of pyrexia in acute gout. If we look into some of the older writers,

we find gout placed amongst the fevers. It has been described as "a tertian fever terminating in fourteen days." This definition was laid down before the days of clinical thermometry, but it may not be quite worthless for all that.

A chart accompanies the card, and it is hoped that the morning and evening temperatures may be recorded until the normal is reached in as many cases as possible.

Attention is directed to the condition of the urine, it being desirable to know more about its characters before, during, and after attacks of acute gout. Thus, free diuresis has been observed to precede an attack in some patients, and many different conditions of the secretion have been noted during and after the attack.

Many other points naturally occur, but it must be borne in mind that this inquiry is a purely therapeutical one, and that it is intended to follow it up at some future time with one of a clinical nature, entering into greater detail upon all the points upon which our knowledge is defective.

The question of treatment, *tuto, cito et jucunde*, is the pre-eminent one for the present. It may be safely assumed that there are many men in possession of the knowledge of prudent and well-tried measures for the relief of acute gout, both local and constitutional; and that their knowledge should be more widely spread is much to be desired.

Many of the ablest practitioners write little. Would that the younger members of the profession could sit at their feet and learn from them some of their hardly acquired art! "Men who can write books, but do not," are rightly termed "valuable," but should not be permitted to carry to the grave any fragments of knowledge that may help to heal the woes of those they leave behind them.

*The Committee is indebted to Dr. DYCE DUCKWORTH for the preparation of this Memorandum.*

## No. VI.

ACUTE GOUT : *especially with reference to its treatment.*

*N.B.—For this purpose, a classical attack, affecting a great toe-joint (true podagra) is intended for observation.*

Observer's Name .....  
Address .....  
Date of last obs.....

(Reply where possible by erasing words on card.)

M. or F. Age.          Occupation.  
Residence.  
Temperate, intemperate, total abstainer. How long?  
Average total amount of fluid taken in 24 hours          pints.\*

\* No. of ozs. or pts. usually taken in 24 hours of—

	Pts.	Ozs.
Wine .....		
Spirits .....		
Malt liquors .....		
Coffee .....		
Tea.....		
Water .....		
Other drinks .....		
Total .....		

N.B.—A tumbler = 9 ozs.  
A claret glass = 3 ozs.  
A sherry glass = 2 ozs.  
A breakfast cup = 8 ozs.

Urine—Quantity.          Increased.          Unaltered.          Diminished.

Before attack.  
During „  
After „

Colour.

Sp. gr.

Reaction.

Albumen.

Sugar.

Deposits.

Before.  
During.  
After.

Digestion.

Appetite.

Thirst.

Pain aft. food.

Acidity.

Flatulence.

Before.  
During.  
After.

Action of Bowels.

Frequency.

Character.

Colour of stools.

Before.  
During.  
After.

Sweating.

Much.

Moderate.

None.

Before.  
During.  
After.

Pain relieved. Date.  
After relief of pain swelling increased, diminished.

Swelling disappeared. Date.

Desquamation of swollen part. Date.

Complete recovery. Date.

Other joints affected.

Treatment.—When begun.

Drugs used.

How often?

How long?

Local.

Large eater, moderate eater, small eater.

Eats meat          times a day.

Any peculiarity in diet.

Strong, moderate, weak. Stout, moderate, spare.

Complexion—florid, medium, pallid.

Hair—dark, intermediate, fair, grey.

Habitual temper—irritable, placid, or

Skin acts much, moderately, little.

Physical exercise—much, moderate, little.

Exciting causes, if any.

First, second, or          attack. Date of first attack.

Date and hour of onset of present attack.

Pain remitting.          Increased at night.

Which great toe affected? Right, left, or both.

Character of Attack.—Rigor severe, moderate, slight, absent.

Pain—severe, moderate, slight.

Redness—bright, dusky, moderate, slight, absent.

Swelling of joint—much, moderate, little.

Oedema—severe, moderate, slight, absent.

Baths. Nature. Temperature. How often.  
Dietetic.  
Symptoms forewarning attack—irascibility, malaise, insomnia, dyspepsia, or other symptoms.  
Subsequent health improved, unaltered, impaired.  
Other ailments to which patient is liable.  
Previous severe illnesses.  
Family history of Gout.  
(This Card as soon as filled up to be returned to Secretary of the Local Sub-Committee.)



## Original Communications.

---

[\* \* The Committee do not hold themselves responsible for any opinions expressed in these Articles.]

---

### CALCULATION OF THE PROBABILITY OF THE ACCIDENTAL AND FATAL INCIDENCE OF PHTHISIS UPON BOTH HUSBAND AND WIFE.\*

BY G. B. LONGSTAFF, M.A., M.B. OXON., M.R.C.P.

*Required*—To find in how many cases in England and Wales during the decade 1871—80 both husband and wife would die of Phthisis, assuming that the chances for married and single are equal in all respects.

Let mean no. of married males, 1871—80 (Census)

$$= M = 4,130,133$$

These are also known for seven ages, viz. :—

15—	5,906	= $\mu_1$
20—	232,332	= $\mu_2$
25—	1,140,049	= $\mu_3$
35—	1,093,663	= $\mu_4$
45—	830,395	= $\mu_5$
55—	528,443	= $\mu_6$
65—	299,345	= $\mu_7$

So that  $M = \mu_1 + \mu_2 + \dots + \mu_7$

\* It occurred to the Secretary of the Committee that it would be possible to calculate the chances that both husband and wife should die of Phthisis, excluding the question of contagion, and he applied to Dr. Longstaff for his assistance. Dr. Longstaff has most kindly afforded the Committee the aid of his experience and ability as a statistician, and has furnished the following calcula-

Similarly mean married females = 4,193,246 = F, showing an excess of wives over husbands = 63,113, which is probably to be explained (a) by husbands on or beyond seas, (b) by deserted wives, the husbands being returned as single. To find the age distribution of the M wives, the census numbers must be adjusted

by multiplying by the factor  $\frac{M}{F} = \kappa = \cdot 984947$ .

Thus:—

15—	33,495	$\times \kappa =$	32,991	$= \phi_1$
20—	381,668	$\times \kappa =$	375,923	$= \phi_2$
25—	1,263,439	$\times \kappa =$	1,244,420	$= \phi_3$
35—	1,093,386	$\times \kappa =$	1,076,927	$= \phi_4$
45—	776,040	$\times \kappa =$	764,358	$= \phi_5$
55—	441,932	$\times \kappa =$	435,280	$= \phi_6$
65—	203,286	$\times \kappa =$	200,226	$= \phi_7$

So that  $M = \kappa F = \phi_1 + \dots + \phi_7$ .

Now we get from the Registrar-General's Reports, 1871-80—

	MALES.				FEMALES.			
	Mean No. living.		Dying of Phthisis.		Mean No. living.		Dying of Phthisis.	
15—	1,176,491	Say $m_1$	19,723	Say $d_1$	1,187,331	Say $f_1$	28,459	Say $\delta_1$
20—	1,032,135	$m_2$	31,927	$d_2$	1,134,358	$f_2$	35,617	$\delta_2$
25—	1,705,568	$m_3$	63,070	$d_3$	1,861,449	$f_3$	65,973	$\delta_3$
35—	1,324,405	$m_4$	54,549	$d_4$	1,431,548	$f_4$	48,663	$\delta_4$
45—	997,999	$m_5$	38,532	$d_5$	1,087,587	$f_5$	26,786	$\delta_5$
55—	681,568	$m_6$	21,774	$d_6$	755,902	$f_6$	13,436	$\delta_6$
65—	512,993	$m_7$	8,015	$d_7$	618,702	$f_7$	5,511	$\delta_7$

If there are  $m$  males living during a certain period, and during same period  $d$  males die of Phthisis, the probability of any one of the  $m$  males dying of it =  $\frac{d}{m}$

But we are dealing with M males, of 7 ages, viz., 15—20 =  $\mu_1$ , 20—25 =  $\mu_2$ , &c., the probabilities of dying of Phthisis at these ages being  $\frac{d_1}{m_1}$ ,  $\frac{d_2}{m_2}$  &c.

tion as a guide for further enquiries. From his results it is easy to ascertain the average number of such cases that should fall under the observation of each practitioner during any given period.

∴ The number of the M males who will probably die of Phthisis within the assigned period is

$$\mu_1 \frac{d_1}{m_1} + \mu_2 \frac{d_2}{m_2} + \dots + \mu_7 \frac{d_7}{m_7} = X.$$

But  $\frac{d_1}{m_1} = .01676427$     Multiplying by  $\mu_1 = 99.01$

$\frac{d_2}{m_2} = .03093282$     „     $\mu_2 = 7,186.68$

$\frac{d_3}{m_3} = .03697884$     „     $\mu_3 = 42,157.64$

$\frac{d_4}{m_4} = .04119859$     „     $\mu_4 = 45,057.38$

$\frac{d_5}{m_5} = .0386093$     „     $\mu_5 = 32,060.97$

$\frac{d_6}{m_6} = .03194669$     „     $\mu_6 = 16,882.12$

$\frac{d_7}{m_7} = .01562406$     „     $\mu_7 = 4,676.98$

Total = X = 148,121,

i.e., *Married males* who will die of Phthisis.

Corresponding to these X husbands are X wives. The number of wives alive at the respective ages may be assumed to be proportional to  $\phi_1, \phi_2, \dots, \phi_7$ , and to form a series  $\chi_1, \chi_2, \dots, \chi_7$

Thus  $\frac{\chi_1}{\phi_1} = \frac{X}{\kappa F} = \frac{X}{M} \therefore \chi_1 = \frac{X}{M} \phi_1$  &c., &c.  $\chi_7 = \frac{X}{M} \phi_7$

$\frac{X}{M} = \frac{148,121}{4,130,133} = .0358635 = \lambda$

$\lambda \phi_1 = \chi_1 = 1,182$

$\lambda \phi_2 = \chi_2 = 13,481$

$\lambda \phi_3 = \chi_3 = 44,629$

$\lambda \phi_4 = \chi_4 = 38,623$

$\lambda \phi_5 = \chi_5 = 27,412$

$\lambda \phi_6 = \chi_6 = 15,610$

$\lambda \phi_7 = \chi_7 = 7,181$

The chances of these X wives (*i.e.* wives of X husbands who



have died of Phthisis) dying of Phthisis within the assigned period will be in like manner—

$$Y = \chi_1 \frac{\delta_1}{f_1} + \chi_2 \frac{\delta_2}{f_2} + \dots + \chi_7 \frac{\delta_7}{f_7}$$

$\chi_1 \delta_1 =$	33,620,805	Dividing by $f_1 =$	28·31
$\chi_2 \delta_2 =$	480,110,731	„ $f_2 =$	423·29
$\chi_3 \delta_3 =$	2,944,325,516	„ $f_3 =$	1,581·73
$\chi_4 \delta_4 =$	1,879,492,254	„ $f_4 =$	1,312·90
$\chi_5 \delta_5 =$	734,264,444	„ $f_5 =$	675·13
$\chi_6 \delta_6 =$	205,826,984	„ $f_6 =$	272·29
$\chi_7 \delta_7 =$	39,575,414	„ $f_7 =$	63·97

$$\text{Total} = Y = 4358$$

The closest approximation to the number required that the data admit of.

*Now for the interval between death of husband and wife.*

To avoid fractions we will suppose 100 couples living. Also that the deaths occur at regular intervals throughout each year.

Let P be the probability that 1 wife (being the widow or wife of a phthisical husband) will die during the 10 years.

$\therefore$  100 P gives the proportion of the 100 wives who will die during the decade, within 10 years of their husbands.

How many wives will die within 9 years of 1st death?

Of those who die in 2nd, 3rd, . . . 9th years (i.e., *ex. hypoth.* 80), the wives may die in *any* of the 10 years.

$\therefore$  the number of these who will probably die is 80 P.

But wives of those dying in 1st and 10th years have only 9·5 years to die in to fulfil the condition.

$\therefore$  the number of these who will probably die is 19 P.

$\therefore$  the whole number who will probably die *within 9 years* = 99 P.

Similarly for 8 years, those in 3rd . . . 8th have full chance = 60 P.

Those in 2nd and 9th years  $9\cdot5 \times 2 = 19$  P.

Those in 1st and 10th years  $8\cdot5 \times 2 = 17$  P.

$\therefore$  the whole number who will probably die *within 8 years* = 96 P.

The whole series will be found to be—

within 10 years = 100 P	within 5 years = 75 P
„ 9 „ = 99 P	„ 4 „ = 64 P
within 8 years = 96 P	„ 3 „ = 51 P
„ 7 „ = 91 P	„ 2 „ = 36 P
„ 6 „ = 84 P	„ 1 „ = 19 P

Or if  $n$  = number of years within which the 2 events are to occur,

$$\left\{ 100 - (10 - n)^2 \right\} P$$

*But of mean population of England and Wales it has been shown that it is probable (assuming no infection) that in*

$$4358 = Y$$

*cases both man and wife would die of phthisis during the 10 years 1871—80.*

∴ in  $\frac{19}{100} Y = 827.98$  cases the 2 events would occur within 1 year of each other.

$\frac{36}{100} Y = 1568.81$	„ 2 „
$\frac{51}{100} Y = 2222.48$	„ 3 „
$\frac{64}{100} Y = 2788.99$	„ 4 „
$\frac{75}{100} Y = 3268.35$	„ 5 „
$\frac{84}{100} Y = 3660.55$	„ 6 „
$\frac{91}{100} Y = 3965.59$	„ 7 „
$\frac{96}{100} Y = 4183.48$	„ 8 „
$\frac{99}{100} Y = 4314.22$	„ 9 „
$\frac{19}{100} Y = 827.98$	cases during first year.

And ∴ in  $\frac{17}{100} Y = 740.83$  cases between 1 and 2 years.

$\frac{15}{100} Y = 653.67$	„ 2 „ 3 „
$\frac{13}{100} Y = 566.51$	„ 3 „ 4 „
$\frac{11}{100} Y = 479.36$	„ 4 „ 5 „
$\frac{9}{100} Y = 392.20$	„ 5 „ 6 „
$\frac{7}{100} Y = 305.04$	„ 6 „ 7 „
$\frac{5}{100} Y = 217.89$	„ 7 „ 8 „
$\frac{3}{100} Y = 130.74$	„ 8 „ 9 „
$\frac{1}{100} Y = 43.40$	„ 9 „ 10 „

The above line of argument holds good equally whether husband or wife die first.

If there be  $N$  medical practitioners in England and Wales, and  $n$  of these send in returns, then (assuming no infection, and assuming that those who send in returns have an average share of practice) they should have *observed* during the ten years a number of cases equal to  $\frac{n}{N}$  of the above numbers, presumably they would *record* a smaller series of numbers. According to the *Medical Directory* for 1883,  $N = 15,462$ . (This number does not include army and navy medical officers, but, on the other hand, it does include many retired medical men.)



## OBSERVATIONS ON THE COLLECTIVE INVESTIGATION OF DISEASE, ESPECIALLY WITH REGARD TO ACUTE PNEUMONIA.

By R. L. BOWLES, M.D., F.R.C.P., FOLKESTONE.

It would seem desirable that in this, the first publication of the Collective Investigation Committee, some account should be taken of the effect of the movement on the minds of practitioners both in London and in the country. It is found that some who are not already members of the Association, desire to become so, that they might take part in the work of Collective Investigation of which they think most highly; others prefer to drift along in their routine, and not be troubled to think unnecessarily; they say, what for? why should I trouble myself? the worry and trouble will never pay! no good will come of it! and advance *laissez faire* arguments of various kinds. Others, again, are deterred from action by diffidence, by modesty, by a fear of showing their ignorance, and most of all by a want of the habit of writing,—a want, *i.e.*, of putting their ideas on paper in an accurate and scientific manner. Then again there are some who *do* think for themselves, who have struggled out of the ruts of routine, and have struck out lines for themselves on a true scientific basis, and who interrogate Nature rather than books; these find it difficult to answer the cards literally enough to satisfy their minds: they find disease does not run in the systematic grooves laid down for it, but that it is often apparently erratic, irregular, undefinable, and influenced by surroundings that are new to our experience, and quite untold of in our books.

From these independent thinkers, Time will soon remove difficulties; they cannot remain long in their troubles, for the very freedom of action they have assumed will direct them in the right

way, and induce them to help our Committee in an active and efficient manner.

It is from these free lances that the real good of the work will come;—for although in the admirable memoranda of guidance drawn up and published with the cards, the issue of the inquiry is narrowed for the purpose of having a limited number of important questions settled with exactness,—these men will soon widen it, and open up fresh matters of interest which will materially help the work of those at the helm and increase the interest of all concerned.

It should be the object of the local committees to develop the frame of mind, just referred to, in all the members, to indicate in what way any observations additional to those required by the card may be dealt with, and to assist in arranging them, so that they may be readily utilized by the committees appointed to collate and tabulate the returns. Further, the local committees or individual members should stimulate the indifferent, encourage the diffident, and offer help and advice to all. It is true this requires tact and delicacy, but a man with a proper sense of the object he has in view, will scarcely fail in that direction; the scientific instinct and the feelings of a gentleman will sufficiently guide him. It is true that the busy man will find it at first irksome to fill up cards; but soon he will see that it is a real help to him, that it directs his attention at once, and in the shortest way, to the points to be observed, and suggests many matters of interest that he would otherwise have lost sight of: and that it early adds—to the routine business of his profession—a scientific and intellectual interest which brings its own pleasure and its own reward.

By increasing and systematizing his knowledge, it gives him confidence and a facility of appreciating fresh cases, which not only save time and mental wear and tear, but favourably impress his client and add to his reputation and success.

This undertaking I foresee will not result alone in the recording of a series of bald facts, such as those demanded by the cards already issued, but will,—by the very difficulties encountered in the attempt to dovetail the symptoms of excited and disturbed Nature into the order of the questions on the card,—stimulate fresh thought, and demonstrate in a practical manner how unsafe it is

to drift into routine, and to expect Nature to conform in her derangements with the dogmas already accepted and propagated by the current medical literature. It was left for the genius of Professor Humphry not only to suggest this really great movement, but also to promote it as he is now doing with much pains and self-denial in a most masterly manner.

In consequence perhaps of the prevalence of catarrhal and chest affections during the severe weather of this early spring, the cards on "Acute Pneumonia" appear to have excited most interest and discussion. Many friends, like myself, were struck, on first attempting to fill them up, at finding it a more difficult task than they had anticipated; our cases would not exactly fit into the order of the card, and it brought to my mind other cases in the past in which irregularities in the course of the disease had developed themselves, rendering both the diagnosis and treatment a matter of some anxiety.

It will perhaps be useful at this stage of our inquiry to draw attention to some of these and, as well, incidentally to comment on the card and the memorandum sent with it for guidance. Experienced physicians and teachers, of course, are well acquainted with the complications, varieties, and irregularities I refer to, but to be successful as teachers they find it desirable to keep these more or less in the background. Teachers must be systematic and dogmatic, and accentuate the ordinary symptoms, in order to impress certain definite principles on the as yet only partially tutored mind.

But when the young practitioner enters into the responsibilities of work he finds that, valuable as his principles may be, he has much to unlearn! He soon discovers that Nature has her eccentricities, and that to deal with them, he must cast aside his dogmas and get his light from her laws, rather than from systems, authorities, and books.

I had been much struck by having at short intervals three cases of asthma in children of about 12 years of age, with emphysematous lungs, who were attacked with pneumonia, but in which the physical signs did not display themselves until late in the disease (the fourth, fifth, or sixth day); it was quite certain that they were not overlooked, for chest inflammation was suspected from the first, and carefully sought for. A boy at school was



seized with feverishness and sickness on November 21st, 1879; on the 23rd the temperature was 106·2, and the pulse 164; these symptoms continued as grave until the 26th (fifth day), when the temperature dropped to 98. On 27th (sixth day) it rose to 105, and dulness and tubular breathing at about the middle of the left lung behind, were noted for the first time with certainty. On 28th (seventh day) the temperature again fell and remained low. Dr. Eastes of Folkestone attended the case, and I saw it in consultation on the third day. It was important to pronounce early on the nature of the case, lest it should be infectious, but although it was impossible to give a positive opinion, a previous experience of the sudden onset of high fever in emphysema, led me to express an opinion in favour of its being an attack of pneumonia.

A young lady with asthma and emphysema was seized with high fever, and on the fifth day the physical signs of pneumonia became apparent, there was here early nerve prostration and a bilious diarrhoea, giving rise to fears of the presence of typhoid fever, especially as on the sixth day the tongue became dry and brown, but on the seventh day the crisis came, and speedy convalescence followed.

Another young lady (Miss M.) of about the same age, and in a similar condition, had a similar attack, and not until the sixth or seventh day of the malady, could pneumonia be diagnosed with certainty, although carefully sought for twice daily.

In all these three cases the temperature was high (from 104 to 106), but the pulse and respiration ratio was unusual, the pulse being very rapid, and the respiration very little above the normal standard; these unusual conditions I believe to belong to the pneumonia of emphysematous lungs, at least in my experience it has been, as far as I can remember, invariably so. I believed too at one time that the late development of the physical signs of pneumonia was consequent *alone* upon emphysematous lungs, but Dr. Tyson of Folkestone encountered a case in which the symptoms were not developed until the seventh day, in which there was *no* emphysema, and since then he has observed in a child of seven, that they did not become apparent until the fifth day. The low typhoid form is not unfrequently late in displaying itself, and the alcoholic form has some peculiarities; but on two occasions I have had difficulty in deciding

whether I should relegate a case to the card on "acute pneumonia," or "acute rheumatism." In one, a boy of 14, in good health and in active exercise, after getting hot, and then sitting on the beach, was taken with stiff neck; the stiffness suddenly passed off, and fever setting in, pneumonia appeared on one side; several days later, as this was subsiding, the same condition arose in the opposite lung; a week later pericarditis and an aortic bruit became evident, there was no marked joint affection, but heavy perspirations from time to time.

Dr. Thomas Eastes has kindly supplied me with notes of a case of this nature, which has recently been under his care.

A lady "caught a cold," upon which flying rheumatic pains in the knees, ankles, and elbows supervened. Ten or twelve days later, after a severe shock to her nervous system, she had some irregular rigors, succeeded by pain in the left shoulder and cardiac region; a pericardial friction sound was detected, and she was put under treatment for rheumatic fever: on the day following, the temperature became very high, and the physical signs of pneumonia were developed on the left side: on the fourth day the right lung was attacked, and this was succeeded by effusion into the right pleura; the crisis was well-marked, and a speedy recovery ensued.

This lady fifteen years previously suffered from a severe attack of pneumonia, preceded by pains of a rheumatic character in the larger joints, and had also from time to time sub-acute rheumatism, and what had been called gouty bronchitis.

When cases of this nature come under our observation it forces upon us the necessity of, in all acute illnesses, taking fully into consideration, not only the immediate "antecedent illness," as demanded on our card, but also the condition antecedent to that state of unrest and derangement of the natural functions, which we term illness: diathesis, heredity, idiosyncrasy, habit, family, and mental, as well as physical, surroundings, and, even more than these, must be entertained before we can explain why the blasting wind which blows on all alike only here and there selects a victim. It is in this direction that so much help may fairly be expected from the family doctor, who alone possesses all the information required to clear up many an obscurity in illness.

In some cases it is not easy to note with certainty the beginning

of the disease. A gentleman in perfect health returned from a walk, meeting a severe east wind for five miles, next day he had "a cold," two days later he travelled to London, and two days later still, was severely ill, the nervous system was profoundly impressed, and there was albumen in the urine. Pneumonia was guessed at, and two days after, the development of the physical signs confirmed the diagnosis. No well-marked rigor or other initial symptom of pneumonia could be elicited to mark the onset of the disease, and this was rendered even more difficult by the fact of everyone else in the house having "bad colds."

In cases similar to those just described, and in all others abnormal, or rather beside and beyond the requirements of the card, I would suggest that the card should be filled up literally, and a very short account of the additional facts appended to it, leaving it to the Committee to utilize them as they think best. In the course of time this additional matter will become of value for guidance in the issuing of fresh cards, enlarging the area, and thus increasing the worth of this great work of the British Medical Association.



## Suggestions for Future Investigations.

---

[\* \* The Committee do not hold themselves responsible for any opinions expressed in these Articles.]

---

### PROPOSAL FOR AN INQUIRY INTO THE ASSOCIATION OF WHOOPING-COUGH WITH MEASLES.

BY W. B. CHEADLE, M.A., M.D., CANTAB., F.R.C.P.

*Physician to St. Mary's Hospital, and to the Hospital for Sick Children,  
Great Ormond Street.*

It has been observed that whooping-cough occurs not unfrequently in association with measles, both as an epidemic and in individuals, and a similar association has been noted with scarlatina and with varicella.

The object of the proposed inquiry is to ascertain, if possible, whether the connection of whooping-cough with these eruptive fevers is a mere coincidence, or occurs with sufficient constancy, order, and regularity to warrant the assumption that a true pathological relation exists between them; a point which seems especially adapted for elucidation by means of collective investigation.

Should the existence of an actual relation between these diseases be established, the further question would arise, whether it was due to the concurrent development of the two forms of contagion from a common source, or the direct origin of one from the other; or to the prevalence of atmospheric or other conditions favourable to the development of both, although generically distinct; or to the influence of one form of contagion upon the individual recipient in creating a predisposition to the other.

These points might form the ground of a more extended inquiry subsequently.

Two cards of inquiry are suggested in the first instance, viz., one, A., for cases of *Whooping-cough*, for the purpose of ascertaining whether any connection can be traced with antecedent or concurrent measles; and another, B., for cases of *Measles*, for the purpose of ascertaining whether, conversely, any connection can be traced with antecedent or concurrent whooping-cough.

A. *Whooping-Cough.*

Patient's name. Age. Sex. Date of attack.

Catarrh. (Conjunctivitis. Redness of fauces. Bronchitis.)

Transient eruption on skin. Complications.

Previous attack of measles, if any.

(1) In patient. Date.

(2) In family. Individuals affected. Date of attack in each.

(3) Epidemic in district. Date.

Form of measles.	Ru- {	<i>Symptoms.</i> (Sneezing. Conjunctivitis. Hoarseness. Laryngeal cough. Croup. Affection of tonsils. Swelling of submaxillary or cervical glands. Bronchitis. Pneumonia.)
Whether genuine beola or Rötheln.		

Presence or absence of albumen in urine.  
Casts.

Character of eruption.

B. *Measles.*

Name. Age. Sex. Date of attack.

Whether genuine Rubeola or Rötheln.	{	<i>Symptoms.</i> (Sneezing. Conjunctivitis. Hoarseness. Laryngeal cough. Croup. Affection of tonsils. Swelling of submaxillary or cervical glands. Bronchitis. Pneumonia.)

Presence or absence of albumen in urine.  
Casts.

Character of eruption.

Previous attack of whooping-cough, if any.

(1) In patient. Date.

(2) In family. Individuals affected. Date of attack in each.

(3) Epidemic in district. Date.

[A series of questions exactly analogous to those suggested in the form for measles could be drawn up for scarlatina and variçella].

PROPOSAL FOR AN INQUIRY CONCERNING  
WHOOPING-COUGH.

BY JUDSON S. BURY, M.D., B.S., LOND.

*Reasons for Inquiry.*—In England there is not, so far as we know, any children's hospital which has beds set apart for cases of whooping-cough. In out-patient practice it is obviously impossible to view the disease intelligently on all sides. It can, then, be thoroughly investigated only by the family practitioner.

Whooping-cough is one of the commonest and one of the most fatal of infantile diseases, yet we know scarcely anything with regard to its ætiology, we are still ignorant as to its essential nature, and are still uncertain whether any drug materially influences the natural course of the disease.

*Objects of Inquiry.*—Some of the main objects of the inquiry would be to ascertain (1) the atmospheric conditions at the onset of the epidemic or of sporadic cases; (2) the recent antecedents, and the relations of whooping-cough to measles and other epidemics; (3) its relations to other convulsive disorders, to tubercle, to deformities of the chest, &c., &c.; (4) to note the common ailments in families where the disease has occurred, and to contrast these with those occurring in families peculiarly exempt from the malady; (5) to make careful observations of unusual cases, such as whooping-cough in the newborn child and in old age; (6) to investigate the influence of drugs, of counter-irritation, &c.—the number of whoops per diem affording a useful criterion; (7) in case of death, to carefully record all morbid appearances, especially, perhaps, the condition of the larynx, bronchial glands, and nervous system.

These and other points suitably worked out as headings on a card, would lead, we believe, to more accurate knowledge, and possibly to better indications for the treatment of this intractable disease.

---



## THERAPEUTICS.

BY W. E. BUCK, M.A., M.D., CANTAB.

*Physician to the Leicester Infirmary and Fever House.*

It is to be hoped that, in the interests of the profession and of the public, the Committee of the Collective Investigation will not limit its researches to the causation and natural history of disease, but will also endeavour to obtain information as to treatment and the action of drugs.

That proper treatment is impossible without correct diagnosis is of course a truism, and it is equally patent that the knowledge of the causation and natural history of a disease is all-important to its therapeutics. These truths have, however, impressed themselves to such an extent upon the minds of some of our more eminent and rising physicians, that, in devoting themselves to diagnosis and pathology, they have neglected treatment, and have come to regard it as almost a form of quackery.

The scepticism and *cui bono* ? of the present day has extended to therapeutics, to the manifest disadvantage of the physician and of his patient, who will probably be thrown into the hands of quacks who will pretend to do for him, and who may possibly do for him, what has not been done by scientific men. As it is, the public place more confidence in the surgeon than in the physician. Surgery with its brilliant results draws away every year more and more young men from the scientific pursuit of medicine. Education in therapeutics is at a low ebb in our great schools of medicine, and the students have to gain a knowledge of treatment in their experience of later years at the expense often of their patients and of their reputation.

Cannot the Committee of Collective Investigation do something to remedy this state of things, and to place therapeutics on a sounder basis ?

General practitioners who have been long in practice, and who have taken an interest in the subject, have been shrewdly acquir-

ing a knowledge of treatment ever since they left their hospitals, and their knowledge generally dies with them. Their interest lies rather in the practical than in the scientific pursuit of their profession; they do not reflect much upon the causation and natural history of the cases they attend, but they watch the effect of certain drugs in certain cases, and base their reputation not on the discoveries they make, but on the cures they accomplish.

If this information could be obtained (and no doubt much could be obtained, for the general practitioner is usually communicative on subjects in which he finds himself at home), and could be systematically and scientifically arranged, it would be of the greatest value, and might lead the way to a systematic use of drugs that would be more comprehensive and satisfactory than anything we can yet hope for. Materials might be collected that would be invaluable in the hands of the able scientific men to whose lot it would fall to examine, classify, and estimate the value of the information received.

The special points to which attention might be drawn might be the value of giving certain drugs in small and frequent doses rather than in large doses at longer intervals; the apparently antagonistic and varied action of certain drugs given in large, as contrasted with the action of the same drugs given in small doses; and the definite action of drugs in various diseases and on various constitutions.

Some valuable drugs are almost going out of use because our students are not taught how to use them, and if men do not know how to employ them they are right to abstain from doing so, as powerful drugs are as dangerous when used by the inexperienced as the knife would be in the hands of an incompetent surgeon.

Some years ago too little attention was paid to diagnosis and pathology, and too much faith was placed in the ignorant use of drugs. The pendulum is now swinging in the opposite direction.

No one can over-estimate the value of correct diagnosis and pathology, the foundation of all medical science, but even diagnosis and pathology are not all-important, and are but a means to an end.

## PROPOSAL FOR AN INQUIRY ON INFECTIOUS SORE THROAT, WITHOUT RASH.

By E. T. WILSON, M.B., OXON., F.R.C.P.

*Physician to the Cheltenham General Hospital.*

AMONG minor matters requiring investigation by the Committee, I would suggest "Infectious Sore Throat, without Rash." It is undoubtedly of frequent occurrence, and is at times without *any* apparent connection with scarlet fever poison. Death is not an infrequent result. A paper read by Dr. Bond, at the Worcester meeting, in August last, on "Scarlatinal Sore Throat and its Relation to other Throat Affections," has a direct bearing on the subject; but a wider inquiry would undoubtedly produce many additional instances and throw some light on the ætiology of a somewhat obscure affection.

Another question worth solving is "under what conditions, if any, acute Follicular Tonsillitis is infectious? and how far its occurrence, or its infectious character, is due to insanitary conditions?"

## SUGGESTIONS FOR AN INQUIRY ON INFLUENZA.

By J. FOSTER PALMER, Esq.

THERE are few diseases concerning which collective investigation appears to promise more fruitful results than influenza. Although so much has been written, it is nevertheless certain that at present but little can be stated with anything like certainty on the subject.

I beg to subjoin a few of the reasons which have led me to suggest the probable importance of a collective inquiry into the external circumstances connected with this disease.

1st. We know of no other affection which is so obviously, or, at any rate, apparently, dependent on meteorological conditions.



All authors on the subject have agreed in connecting catarrhal epidemics with cold, moist seasons, and sudden atmospheric vicissitudes (*vide* Bascombe: "History of Epidemic Pestilences from the Earliest Ages," page 161).

2ndly. Influenza, it is supposed, often leads to more serious manifestations if neglected. The question to be solved here is this:—Will influenza run its course independent of conditions, or is it liable to be artificially cultivated, or to assume grave inflammatory complications outside its original limits of duration and locality?

3rdly. Many of the great pestilential outbreaks of the world have been preceded by epidemics of influenza. This was the case with the great cholera epidemic of 1817, as well as with many others at different periods; in fact, during the last century the two diseases travelled over almost precisely the same ground. The same fact has been observed in smaller outbreaks of disease, the first few cases being diagnosed as influenza, the later ones as some specific fever; it is then usual to class all the cases together, but it is not improbable that both diagnoses may be correct, and that influenza may have preceded the fever. This is a point which a collective inquiry would assist in clearing up. Definite statistics might thus furnish us with most valuable warnings of coming outbreaks, and may also tend to demonstrate the truth or fallacy of the theory which asserts the *progressive development* of zymotic disease.

The special points to which it is desirable that attention should be directed by the questions put on this subject are as follows:—

Temperature.	Sanitary conditions.
Pulse.	Soil and locality.
Physical chest signs.	Meteorological conditions: damp, dry, hot, cold, height of barometer.
Duration of ditto.	State of electric tension.
Coryza.	Direction and force of wind.
Pain in head.	State of health (general).
„ „ eyes.	„ „ (immediately preceding).
„ „ nose.	„ digestive organs preceding.
Muscular pains.	Premonitory symptoms.
Tongue.	Complications, duration.
Appetite.	Sequelæ.
Vomiting.	
Prostration.	
State of digestive organs.	Concurrent { Preceding.
Sporadic or epidemic.	epidemics. { Simultaneous.
	{ Succeeding.

Having thus attempted to trace Influenza in its onward progress, it would be well if we could also trace it backwards to find its earliest manifestations. These we should expect to find, if anywhere, in the simplest form of ordinary head-cold. Here, then, we must commence our observations. The point on which it will be necessary for us to concentrate our attention is this: Does there exist an unbroken line of descent commencing in a mild form of common catarrh, extending to the severer forms of influenza, and thence continued onwards to higher forms of pathological states? To answer this question we must first ascertain whether there are any forms of catarrh which are absolutely non-contagious; and, secondly, whether the milder forms may be developed, either in the same individual or in others, into forms more severe; in the former case by ill-adapted treatment, neglect, or over-fatigue, and in the latter by being reproduced in persons better fitted for its cultivation by reason of some deficiency, temporary or permanent, of the vital powers.

The question of contagion being still *sub judice*, the only practical division that can be made is between catarrh with constitutional symptoms (influenza), and catarrh without (local catarrh). This, however, is only a relative distinction, as infinite variety exists both in the degree of severity and in that of constitutional sympathy, while probably no catarrh is entirely local. It is at present the opinion of most observers that the severity of an attack of influenza depends almost entirely on the previous condition of the patient: there can be but little doubt that this applies to all forms of catarrh, and marked differences have been observed in the same individual attacked during health and during convalescence from acute disease. What is purely local catarrh in the one case becomes typical influenza in the other. My own observations lead me to believe that there is no absolute line of demarcation: that all cases are to some extent contagious, and to some extent constitutional; and that, while no therapeutic measures will cut short or diminish an attack of catarrh, it is not difficult by similar means greatly to increase its severity. These are points to be decided by combined inquiry.

I trust I have adduced sufficient grounds in favour of the attempt thus to trace the development of Influenza in both directions, and to show that the results of such an attempt, whatever

conclusions they may lead to, will not be insignificant. May it not even be possible that we shall be able to trace a continuous development of disease similar to the evolution of animal and vegetable life, commencing in a common cold in the head, and ending in the Plague, which, on more than one occasion, has carried off half the population of the world? Time and patient observation alone will tell.

The questions requiring to be answered in elucidating this portion of the subject would be as follows:—

- |  |   |
|--|---|
| Is present attack attributable to contagion or infection from another patient suffering from—            | } Influenza ?<br>Local catarrh ?          |
| Total number of inhabitants of house.  |   |
| Number of ditto who have, during the fortnight preceeding commencement of present attack, suffered from— | } Influenza ?<br>Local catarrh ?          |
| Date of commencement of each attack.   |   |
| Number of ditto who, during the three weeks following commencement of present attack, suffered from—     | } Influenza ?<br>Local catarrh ?          |
| Date of commencement of each attack.   |   |
| Did the first case which occurred in the house assume the character of influenza or of local catarrh ?   |   |
| Was the first case introduced by the arrival of some fresh person in the house ?                         |   |
| Yes or No.   |   |
| Apparent origin of first case. By contagion or otherwise ?   |   |
| Previous attacks of influenza. Number and dates.   |   |
| Ditto ditto of local catarrh—  | { Approximate number.<br>Few or numerous. |
| State of general health preceeding former attacks of—  |   |
| as—In perfect health.  | { Influenza.<br>Local catarrh.            |
| Same as before present attack.   |   |
| During convalescence from acute disease.   |   |



# Organisation for the Collective Investigation of Disease.

---

## COLLECTIVE INVESTIGATION COMMITTEE OF THE ASSOCIATION.

Professor Humphry, F.R.S. (Chairman); C. G. Wheelhouse, F.R.C.S. (President of Council); W. F. Wade, M.D. (Treasurer); R. L. Bowles, M.D.; A. Carpenter, M.D.; B. Foster, M.D.; C. Macnamara, F.R.C.S.; A. Ransome, M.D.; E. H. Sieveking, M.D.; F. A. Mahomed, M.B. (Secretary).

---

## GENERAL COMMITTEE.

Thomas Aitken, M.D.; Thomas Barlow, M.D.; H. T. Butlin, F.R.C.S.; T. Lauder Brunton, M.D.; W. E. Buck, M.D.; J. Cavafy, M.D.; W. B. Cheadle, M.D.; Sidney Compland, M.D.; J. Ward Cousins, M.D.; A. Davidson, M.D.; N. Davies-Colley, F.R.C.S.; Dyce Duckworth, M.D.; G. Eastes, M.B.; F. Galton, F.R.S.; J. F. Goodhart, M.D.; T. H. Green, M.D.; W. C. Grigg, M.D.; J. W. Howard, F.R.C.S.; J. Hutcheson, F.R.C.S.; G. B. Longstaff, M.B.; Sir W. MacCormac, Bart., F.R.C.S.; Stephen Mackenzie, M.D.; Withers Moore, M.D.; Shirley F. Murphy, Esq.; H. Page, F.R.C.S.; C. Palmer, Esq.; A. Parsons, M.D.; Rees Phillips, M.D.; S. J. Sharkey, M.B.; Octavius Sturges, M.D.; Fred. Taylor, M.D.; C. Turner, M.D.; W. J. Tyson, M.D.; J. Burney Yeo, M.D.; and the local Hon. Secs., who are *ex officio* members of this Committee.

---

## LIST OF LOCAL COMMITTEES IN THE BRANCHES,

*With Returns sent, up to June 30, 1883.*

### Aberdeen, Banff, and Kincardine Branch.

*Committee.*—Angus Fraser, M.D.; R. J. Garden, M.D.; A. Macgregor, M.B.

*Hon. Sec.*—J. Mackenzie Booth, M.D., Union Street, Aberdeen.

*\*Returns.*—I., 10; II., 3; III., 3. Total, 16.

Total returns from the Branch, 16.

### Bath and Bristol Branch.

BATH DISTRICT.—*Committee.*—T. Cole, M.D.; E. Field, M.B.; R. S. Fowler, Esq.; A. E. W. Fox, M.B.; F. Parsons, Esq.; J. K. Spender, M.D.; Alexander Waugh, Esq.

*Hon. Sec.*—R. J. H. Scott, Esq., 13, Bladud Buildings, Bath.

*Returns.*—I., 11; II., 3; III., 4; V., 1; total, 19.

BRISTOL DISTRICT.—*Committee.*—D. Davies, Esq.; N. Dobson, Esq.; E. L.

\* I. Pneumonia; II., Chorea; III. Rheumatism; IV., Diphtheria; V., Syphilis.

Fox, M.D.; A. J. Harrison, M.B.; W. H. Harsant, Esq.; G. F. Rossiter, M.B.; J. G. Smith, M.D.; R. S. Smith, M.D.

*Hon. Sec.*—E. Markham Skerritt, M.D., Richmond Hill, Clifton.

*Returns.*—I., 7; II., 9; III., 9; IV., 1. Total, 26.

Total returns from the Branch, 45.

### Birmingham and Midland Counties Branch.

*Chairman.*—B. Foster, M.D.

*Committee.*—M. H. C. Atkinson, M.D.; G. F. Blake, Esq.; H. W. L. Browne, Esq.; A. H. Carter, M.D.; A. Cunningham, M.B.; C. Dukes, M.D.; W. C. Garman, Esq.; G. W. Homan, Esq.; J. Holmes Joy, M.D.; W. Lattey, Esq.; C. A. McMunn, M.D.; F. E. Manby, F.R.C.S.; R. Mears, Esq.; M. A. Messiter, Esq.; Milner M. Moore, M.D.; J. J. Nason, M.B.; G. A. Phillips, Esq.; E. Rickards, M.B.; J. Sawyer, M.D.; S. J. Smith, M.D.; F. Thorne, Esq.; T. W. Thursfield, M.D.; John Tibbits, M.D.; A. Underhill, M.D.; T. E. Underhill, M.B.; W. F. Wade, M.D.

*Hon. Sec.*—Robert Saundby, M.D., 25, Newhall Street, Birmingham.

*Returns.*—I., 20; II., 12; III., 32; IV., 11. Total, 75.

Total returns from the Branch, 75.

### Border Counties Branch.

*Committee.*—Henry Barnes, M.D.; W. D'O. Grange, M.D.; W. M. Taylor, M.D.

*Hon. Sec. Northern District.*—Vacant.

*Returns.*—I., 1. Total, 1.

*Hon. Sec. Southern District.*—Acting *pro tem.*, Henry Barnes, M.D., Portland Square, Carlisle.

*Returns.*—I., 4; II., 1; III., 4; IV., 5. Total, 14.

Total returns from the Branch, 15.

### Cambridge and Huntingdon Branch.

*Committee.*—William Armistead, M.B.; D. B. Balding, Esq.; T. B. Bradbury, Esq.; J. Bridger, Esq.; E. Bury, M.D.; J. Carter, Esq.; W. H. Copley, Esq.; W. Easby, M.D.; C. N. Elliott, M.B.; William Groom, Esq.; W. R. Grove, M.D.; E. Hasle, Esq.; C. F. Hodson, Esq.; G. M. Humphry, M.D., F.R.S.; R. N. Ingle, M.D.; H. Lucas, Esq.; Alex. W. Smith, M.B.; H. Stear; J. J. Walker, M.D.

*Hon. Sec.*—B. Anningson, M.D., Barton Road, Cambridge.

*Returns.*—I., 14; II., 4; III., 7; IV., 4. Total, 29.

Total returns from the Branch, 29.

### Dublin Branch.

*President.*—John T. Banks, M.D.

*President Elect.*—Edward Hamilton, M.D.

*Vice-Presidents.*—Loombe Atthill, M.D.; Ed. H. Bennett, M.D.

*Council.*—Thomas Darby, F.R.C.S.; Samuel Gordon, M.D.; Thomas W. Grimshaw, M.D.; J. William Moore, M.D.; E. D. Mapother, M.D.; Robert McDonnell, M.D., F.R.S.; P. C. Smyly, M.D.

*Committee.*—C. B. Ball, M.D.; J. H. Benson, M.D.; J. Beatty, F.R.C.S.; Robert Brown, M.D.; John H. Chapman, M.D.; A. H. Corley, M.D.; J. P. Doyle, Esq.; J. M. Finny, M.D.; A. W. Foot, M.D.; William Frazer, M.D.; Christopher Gunn, M.D.; D. H. Hadden, Esq.; Rev. S. Haughton, M.D.; R. A. Hayes, M.D.; A. N. Montgomery, Esq.; William Moore, M.D.; W. C. Neville, M.B.; C. J. Nixon, M.B.; J. F. Pollock, M.D.; John B. Power, Esq.; F. J. B. Quinlan, M.D.; W. G. Smith, M.D.; Thornley Stoker, M.D.; William Stokes, M.D.; H. R. Swanzy, M.D.; H. J. C. Tweedy, M.D.; J. W. Usher, Esq.; J. Vance, Esq.; P. L. Whistler, M.D.

*Hon. Sec.*—George F. Duffey, M.D., Fitzwilliam Place, Dublin.

*Returns.*—I., 5; II., 3; III., 6. Total, 14.

Total returns from the Branch, 14.

**East Anglian Branch.**

*Committee.*—T. E. Amyot, F.R.C.S.; E. G. Barnes, M.D.; H. J. Benham, M.D.; M. Beverley, M.D.; W. Cadge, F.R.C.S.; T. W. Crosse, F.R.C.S.; W. B. Crowfoot, F.R.C.S.; P. Eade, M.D.; G. Elliston, Esq.; J. Lowe, M.D.; C. Palmer, Esq.; F. Seymour, Esq.

*Hon. Sec. for Norfolk.*—S. H. Burton, M.B., St. Giles Street, Norwich.

*Hon. Sec. for Suffolk.*—W. A. Elliston, M.D., St. Peter's Street, Ipswich.

*Returns.*—Norfolk: I., 9; IV., 11. Total 20, with a report of an epidemic of 70 cases, by W. L. King, Esq.

*Returns.*—Suffolk: I., 2; II., 3; III., 2; IV., 19. Total, 26.

Total returns from the Branch, 46.

**East York and North Lincoln Branch.**

*Committee.*—A. Allen, M.D.; J. M. Bramwell, M.B.; Owen Daly, M.D.; G. F. Elliott, M.D.; E. P. Hardey, Esq.; A. Jackson, Esq.; J. F. Nicholson, M.D.; R. H. B. Nicholson, Esq.; J. Sherburn, M.B.; J. E. Smith, M.D.

*Hon. Sec.*—E. O. Daly, M.B., 26, Albion Street, Hull.

*Returns.*—I., 1. Total, 1.

Total returns from the Branch, 1.

**Edinburgh Branch.**

*Chairman.*—D. J. Brakenridge, M.D.

*Committee.*—W. Badger, M.B.; G. A. Berry, M.B.; J. Bishop, M.D.; B. Bramwell, M.D.; J. Carmichael, M.D.; A. L. Currer, M.B.; C. E. Douglas, M.D.; A. Drysdale, M.B.; W. A. Finley, M.D.; J. Foulis, M.D.; A. James, M.D.; D. B. Hart, M.D.; G. Hunter, M.D.; J. Hunter, M.B.; R. Kirk, M.D.; P. McBride, M.D.; A. Matthew, F.R.C.S.; J. W. Moir, M.D.; A. D. L. Napier, M.D.; T. G. Nasmyth, M.B.; J. B. Ronaldson, Esq.; F. R. Ronaldson, M.B.; J. R. Scott, M.B.; R. Spence, M.B.; C. E. Underhill, M.B.; P. A. Young, M.D.

*Hon. Sec.*—G. A. Gibson, M.D., D.Sc., 1, Randolph's Cliff, Edinburgh.

*Returns.*—I., 13; II., 10; III., 7; IV., 2. Total, 32.

Total returns from the Branch, 32.

**Glasgow and West of Scotland Branch.**

*Committee.*—G. Buchanan, M.D.; J. Christie, M.D.; J. Coats, M.D.; W. Frew, M.B.; W. T. Gairdner, M.D.; B. Goff, M.D.; T. Brown Henderson, M.D.; A. L. Kelly, M.D.; W. J. Marshall, M.D.; D. Yellowlees, M.D.

*Convener.*—A. Napier, M.D., 3, Royal Terrace, Crosshill, Glasgow.

*Returns.*—I., 21; II., 4; III., 14; IV., 6. Total, 45.

Total returns from the Branch, 45.

**Gloucestershire Branch.**

*Chairman.*—President of the Branch.

*Committee* of the whole Branch.

*Hon. Sec. for Cheltenham District.*—E. T. Wilson, M.B., F.R.C.P., Westal Cheltenham.

*Hon. Sec. for Gloucester District.*—F. T. Bond, M.D. Montpellier Place, Gloucester.

*Returns.*—Cheltenham District: I., 8; II., 3; III., 3; IV., 2. Total, 16.

*Returns.*—Gloucester District: I., 2; III., 1; IV., 3. Total, 6.

Total returns from the Branch, 22.

**Lancashire and Cheshire Branch.**

**BOLTON DISTRICT.**—*President:* Charles Rothwell, Esq. *Vice-President:* F. B. Mallet, M.D.

*Committee.*—J. J. Barnes, Esq.; J. Barr, Esq.; A. Cosgreave, Esq.; B. Derham, M.D.; C. Glasier, M.D.; G. Howarth, Esq.; J. Johnston, M.D.; J. A. Mackenzie,



M.B.C.M.; R. J. Martin, F.R.C.S.; W. Y. Martin, F.R.C.S.; R. Patrick, Esq.; J. E. Scowercroft, F.R.C.S.

*Hon. Sec.*—De Vere Hunt, Esq., 46, St. George's Terrace, Bolton.

*Returns.*—I., 14; II., 3; III., 7; IV., 2. Total, 26.

LIVERPOOL DISTRICT.—*Committee.*—J. Barr, M.D.; A. Barron, M.B.; W. Macfie Campbell, M.D.; W. Carter, M.D.; A. Davidson, M.D.; F. Pollard, M.D.; A. C. Rich, M.B.

*Hon. Sec.*—F. T. Paul, Esq., 44, Rodney Street, Liverpool.

*Returns.*—I., 16; II., 9; III., 10; IV., 4; V., 6. Total, 45.

MANCHESTER DISTRICT.—*Committee.*—H. Ashby, M.D.; R. Beales, M.D.; J. Dreschfeld, M.D.; T. W. H. Garstang, Esq.; A. Hamilton, Esq.; J. A. Harris, M.D.; H. R. Hutton, M.B.; D. J. Lush, M.D.; S. A. McGowan, M.D.; D. J. Mackenzie, M.D.; J. Mackenzie, M.B.; H. Colley March, M.D.; J. Parks, Esq.; J. B. Perrin, Esq.; A. Ransome, M.D.; C. J. Reushaw, M.D.; W. Roberts, M.D., F.R.S.; C. D. Shepherd, Esq.; J. Starkey Smith, M.B.; G. Thomson, M.D.

*Hon. Sec.*—J. S. Bury, M.D., 36, Fitzwarren Street, Pendleton, Manchester.

*Returns.*—I., 32; II., 13; III., 18; IV., 26; V., 1. Total, 90.

Total returns from the Branch, 161.

### Metropolitan Counties Branch.

*Committee.*—C. A. Aikin, Esq.; W. H. Allchin, M.B.; J. Althaus, M.D.; G. W. Armstrong, Esq.; J. Baber, M.D.; G. H. Bailey, Esq.; E. Ballard, M.D.; J. Wickham Barnes, Esq.; J. G. Barratt, M.D.; Fletcher Beach, M.B.; A. Hughes Bennett, M.D.; G. H. Bishop, Esq.; C. Y. Biss, M.B.; Percy Boulton, M.D.; John M. Bright, M.D.; George D. Brown, Esq.; G. H. Cable, Esq.; W. Watson Cheyne, Esq.; W. Fairlie Clarke, M.D.; W. F. Cleveland, M.D.; J. B. Curgenven, Esq.; W. H. Day, M.D.; Maurice Davis, M.D.; Stamford Felce, Esq.; A. Forsyth, M.D.; F. J. Gant, Esq.; J. J. Gawith, Esq.; J. Goodechild, Esq.; A. Grant, M.D.; G. Hastings, M.D.; W. B. Hemming, Esq.; Charles H. Hill, M.D.; J. W. Hulke, Esq.; F. W. Humphreys, Esq.; W. B. Johnston, M.D.; Norman Kerr, M.D.; J. C. Langmore, M.B.; H. Cripps Lawrence, Esq.; J. T. N. Lipscomb, M.D.; Robert H. Lloyd, M.D.; T. J. MacLagan, M.D.; H. M. Madge, M.D.; H. C. Martin, M.D.; J. J. Merriman, Esq.; W. J. Mickle, M.D.; G. Mickley, M.B.; A. B. R. Myers, Esq.; Robert J. W. Oswald, Esq.; A. Perigal, M.D.; R. H. Prior, M.D.; Walter Rigden, Esq.; G. H. Savage, M.D.; S. W. Sibley, Esq.; W. Squire, M.D.; J. A. Tapson, Esq.; C. Meymott Tidy, M.B.; Godwin W. Timms, M.D.; Morris Tonge, M.D.; T. S. Townsend, Esq.; Edgecombe Venning, Esq.; W. Verdon, Esq.; F. Warner, M.D.; John Way, M.D.; H. Wotton, M.D.; F. J. Wright, Esq.; *ex-officio* members, T. Bridgwater, M. B. (*President of the Branch*); Charles J. Hare, M.D. (*President Elect*); Walter Dickson, M.D. (*Treasurer*); Alexander Henry, M.D.; W. C. Grigg, M.D. (*Hon. Secs. of the Branch*); F. Wallace, Esq.; G. W. Potter, M.D.; R. E. Carrington, M.D.; E. H. Vincen, M.D.; Ridgway Lloyd, Esq. (*District Hon. Secs.*)

*Hon. Sec.*—George Eastes, M.B., 69, Connaught Street, Hyde Park Place, W.

The District Secretaries for the Branch act also as District Secretaries for the Committee.

*Returns.*—I., 21; II., 11; III., 8; IV., 19; V., 1. Total, 60.

Total returns from the Branch, 60.

### Midland Branch.

DERBY DISTRICT.—*Chairman*—W. Ogle, M.D.

*Committee.*—J. W. Baker, Esq.; E. Gaylor, Esq.; T. J. Gentles, Esq.; C. A. Greaves, M.B.; T. Heighton, Esq.; C. H. Hough, Esq.; J. A. Hunt, Esq.; J. Knox, M.D.; W. Legge, Esq.; J. M. Lindsay, M.D.; Duncan Mackenzie, M.D.; C. H. Rowbotham, Esq.; J. A. Sharp, Esq.; W. Webb, M.D.

*Hon. Sec.*—R. Stanley Taylor, M.A., M.B., Friargate, Derby.

*Returns.*—I., 2; II., 5; III., 4. Total, 11.

LEICESTER DISTRICT.—

*Hon. Sec.*—W. E. Buck, M.D., Welford Road, Leicester.

*Returns.*—0.

LINCOLN DISTRICT.—*Committee*.—A. Mercer Adam, M.D.; W. Hamilton Allen, Esq.; A. E. Boulton, Esq.; A. Campbell, Esq.; W. J. Cant, Esq.; W. A. Carline, M.D.; J. T. Collier, M.D.; J. Hadden, M.D.; F. Fawsett, M.D.; R. Lanphier, M.B.; W. Newman, M.D.; W. O'Neill, M.D.; W. J. Pilcher, F.R.C.S.; Ed. Reekitt, Esq.; H. Rainbird, Esq.; G. Mitchinson, Esq.; T. Sympton, F.R.C.S.; F. Sutton, Esq.; T. M. Wilkinson.

*Hon. Sec.*—C. Harrison, M.D., 30, Newland Lincoln.

*Returns*.—I., 4; II., 10; III., 9; IV., 2. Total, 25.

NOTTINGHAM DISTRICT.—*Chairman*.—W. H. Ransome, Esq., M.D., F.R.S.

*Committee*.—J. Beddard, F.R.C.S.; J. O. Brookhouse, M.D.; Thos. Browne, Esq.; C. H. Cattle, M.D.; H. Hatherley, Esq.; L. W. Marshall, M.D.; W. W. Morris, Esq.; F. R. Mntch, Esq.; G. E. Power, Esq.; E. Seaton, M.D.; G. E. Stanger, Esq.; Appleby Stephenson, M.D.; A. Claude Taylor, M.D.; H. O. Taylor, M.D.; C. Haydo White, Esq.; G. B. White, Esq.; J. White, F.R.C.S.; T. Wright, M.D.

*Hon. Sec.*—H. Handford, M.D., General Hospital, Nottingham.

*Returns*.—I., 5; II., 17; III., 7; IV., 6. Total, 35.

Total returns from the Branch, 71.

### North of England Branch.

*Committee*.—R. Anderson, M.D.; T. W. Barron, M.D.; S. W. Broadbent, Esq.; W. Gowans, Esq.; J. Munro, M.D.; G. H. Philipson, M.D.; G. E. Williamson, F.R.C.S.

*Hon. Sec.*—D. Drummond, M.D., Saville Place, Newcastle.

*Returns*.—I., 10; II., 9; III., 6; IV., 7; V., 5. Total, 37.

Total returns from the Branch, 37.

### North of Ireland.

*Committee*.—The Council of the Branch.

*Hon. Sec.*—A. Dempsey, M.D., 26, Clifton Street, Belfast.

*Returns*.—I., 7; II., 9; III., 5. Total, 21.

Total returns from the Branch, 21.

### North Wales Branch.

*Committee*.—S. Griffith, M.D.; R. Hughes, Esq.; J. E. Jones, M.D.; R. O. Jones, Esq.; T. Eyton-Jones, M.D.; A. Eyton-Lloyd, Esq.; Hugh Rees, Esq.; J. Richards, Esq.; R. Roberts, Esq.; A. E. Turnour, M.D.

*Hon. Sec.*—W. Jones-Morris, Esq., Portmadoc.

*Returns*.—I., 11; II., 1; III., 8; IV., 1. Total, 21.

Total returns from the Branch, 21.

### Northern Counties of Scotland.

EASTERN DISTRICT.—

*Hon. Sec.*—J. W. Norris Mackay, M.D., The Tower, 103, High Street, Elgin.

*Returns*.—I., 1; III., 2. Total, 3.

WESTERN DISTRICT.—*President*, T. Aitken, M.D.

*Vice-President*.—W. Bruce, M.D.

*Committee*.—J. Adam, M.B.; R. Craig, M.D.; B. Cruickshank, M.D.; A. Finlayson, Esq.; J. Grigor, M.D.; W. Kennedy, M.D.; R. McCallum, Esq.; D. S. McDonald, M.B.; W. McDonald, M.D.; D. McFadyen, Esq.; F. M. Mackenzie, Esq.; A. R. Mackenzie, M.D.; D. McIntyre, M.D.; J. McNee, M.D.; C. McPherson, M.B.; J. Murray, M.D.; J. J. Ross, M.D.; J. Simpson, M.D.; A. Sutherland, M.B.; J. Vass, M.D.

*Hon. Sec.*—Ogilvie Grant, M.B., 35, Church Street, Inverness.

*Returns*.—I., 3; II., 5; III., 8. Total 16.

Total returns from the Branch, 19.

### Reading Branch.

*Committee.*—J. Harrison, F.R.C.S.; O. C. Maurice, Esq.; G. May, F.R.C.S.; H. H. Phillips, M.D.; W.A.S. Royds, Esq.; J. Shea, M.D.; T. L. Walford, Esq.; E. Wells, M.D.; W. T. G. Woodforde, M.D.; W. B. Young, Esq.

*Hon. Sec.*—R. C. Shettle, M.D., 73, London Street, Reading.

*Returns.*—I., 1; II., 1; IV., 2. Total, 4.

Total returns from the Branch, 4.

### Shropshire and Mid-Wales Branch.

*Committee.*—S. Andrew, M.D.; W. Calwell, Esq.; C. A. Corko, Esq.; E. Cureton, Esq.; W. Bowen Davies; A. Eddowes, M.D.; W. Eddowes, Esq.; H. N. Edwards, Esq.; J. Gill, Esq.; C. H. Gwyuu, M.B.; S. Tayleur Gwynn, M.D.; J. D. Harries, Esq.; T. Morgan, Esq.; A. Strauge, M.D.; E. Tredinnick, Esq.; R. Wilding, Esq.; R. W. O. Withers, Esq.

*Hon. Sec.*—E. S. Scott, M.B., Swau Hill, Shrewsbury.

*Returns.*—I., 27; II., 3; III., 17; IV., 5. Total, 52.

Total returns from the Branch, 52.

### South-Eastern Branch.

**EAST KENT DISTRICT.**—*Committee.*—R. L. Bowles, M.D.; T. Eastes, M.D.; E. Garraway, Esq.; H. A. Gogarty, M.D.; J. Hackney, Esq.; R. Hicks, Esq.; A. Long, Esq.; A. G. Osborn, Esq.; C. Parsons, M.D.; T. F. Raven, Esq.; J. Reid, F.R.C.S.; G. Rigden, Esq.; M. K. Robinson, M.D.; T. S. Rowe, M.D.; W. K. Treves, F.R.C.S.; E. W. Thurston, Esq.; W. J. Tyson, M.D.; F. Wachter, Esq.; G. Wilks, M.B.; S. Woodman, F.R.C.S.

*Hon. Sec.*—T. Whitehead Reid, F.R.C.P., 34, St. George's Place, Canterbury.

*Returns.*—I., 22; II., 3; III., 9; IV., 4. Total, 38.

**WEST KENT DISTRICT.**—*Committee.*—E. Ground, M.B.; A. H. Hallowes, Esq.; C. E. Hoar, M.D.; W. Shaw, M.B.

*Hon. Sec.*—C. Boyce, M.B., 3, Clarendon Place, Maidstone.

*Returns.*—I., 6; III., 3. Total, 9.

**EAST SURREY DISTRICT.**—*Hon. Sec.*—John H. Galton, M.D., Woodside, Anerley Road, Norwood, S.E.

*Returns.*—I., 3; II., 3; III., 4. Total, 10.

**WEST SURREY DISTRICT.**—*Committee.*—E. J. Barker, M.D.; T. M. Butler, Esq.; C. W. Chaldecot, Esq.; S. Crompton, M.D.; W. C. Daniel, M.D.; T. T. Hopcroft, Esq.; A. W. Leachman, M.D.; J. A. Lorrimer, Esq.; J. Morton, M.B.; A. A. Napper, Esq.; T. J. Schollick, Esq.; C. J. Sells, Esq.; S. G. Sloman, Esq., Jun.; J. R. Stedman, M.D.; H. S. Taylor, Esq.; W. W. Young, M.D.

*Hon. Sec.*—T. F. Pearse, M.D., Haslemere.

*Returns.*—I., 20; II., 5; III., 6; IV., 10; V., 2. Total, 43.

**EAST SUSSEX DISTRICT.**—*Committee.*—F. Bagshawe, M.D.; N. P. Blaker, Esq.; J. Ewart, M.D.; R. Graveley, Esq.; C. N. Hayman, M.B.; B. Marsack, Esq.; W. Withers Moore, M.D.; G. J. M. Smith, M.B.

*Hon. Sec.*—J. C. Uhthoff, M.D., 46, Western Road, Hove, Brighton.

*Returns.*—I., 5; II., 5; III., 4; IV., 3. Total, 17.

**WEST SUSSEX DISTRICT.**—*Committee.*—E. I. Bostock, Esq.; L. Buckell, M.D.; T. Fuller, M.D.; W. J. Harris, Esq.; C. Kelly, M.D.

*Hon. Sec.*—G. B. Collet, Esq., 5, The Steyne, Worthing.

*Returns.*—I., 5; II., 1; III., 6; IV., 2; V., 1. Total, 15.

Total returns from the Branch, 132.

### South Midland Branch.

*Committee.*—F. Buszard, M.D.; G. P. Goldsmith, M.D.; R. H. Kinsey, Esq.; G. H. Percival, M.B.; T. More, M.D.

*Hon. Sec.*—G. F. Kirby-Smith, Esq., Northampton.

*Returns.*—I., 2; II., 7; III., 8. Total, 17.

Total returns from the Branch, 17.



**South of Ireland Branch.**

*Committee.*—T. E. Atkins, Esq.; Parsons Berry, Esq.; H. Corby, M.D.; P. J. Cremen, M.D.; P. J. Golding, M.D.; N. Grattan, Esq.; C. A. Harvey, M.D.; T. B. Moriarty, M.D.; Denis C. O'Connor, Sen., M.D., LL.D.; D. B. O'Flynn, M.D.

*Hon. Sec.*—T. Gelston Atkins, M.D., 17, St. Patriek's Hill, Cork.

*Returns.*—I., 4; II., 1; III., 1; IV., 1. Total, 7.

Total returns from the Branch, 7.

**South Wales and Monmouthshire Branch.**

*Committee.*—S. E. Bligh, M.B.; G. A. Brown, Esq.; G. A. Davies, Esq., Newport; H. N. Davies, Esq., Cymner; W. H. Davies, Esq., Cwm Dow House; A. P. Fiddian, M.B.; J. Farrant Fry, Esq.; Evan Jones, Esq., Aberdare; J. Talfourd Jones, M.B., Brecon; J. W. Mulligan, M.D.; W. Price, M.B.; J. L. W. Ward, Esq.; T. J. Webster, Esq.; D. J. Williams, Esq., Llanelly.

*Hon. Secs.*—D. A. Davies, M.B.; De la Beeche Street, Swansea; A. Sheen, M.D., Halswell House, Cardiff.

*Returns.*—I., 33; II., 16; III., 8; IV., 3; V., 2. Total, 62.

Total returns from the Branch, 62.

**South-Western Branch.**

*Committee.*—E. J. Adkins, Esq.; C. Aldridge, M.D.; E. S. Angore, Esq.; L. Armstrong, Esq.; A. de W. Baker, Esq.; J. Blamey, Esq.; T. Boyle, Esq.; C. Bulteel, Esq.; C. W. Chubb, Esq.; H. G. Cumming, Esq.; W. H. Dodge, Esq.; J. W. Gill, Esq.; H. Greenway, Esq.; H. Harden, Esq.; J. Harper, Esq.; J. D. Harris, Esq.; J. U. Huxley, M.D.; G. Jackson, Esq.; P. Q. Karkeek, Esq.; J. Kempthorne, Esq.; A. W. Kempe, Esq.; G. Kerswill, Esq.; D. H. King, Esq.; E. C. Langford, Esq.; H. de Legh, Esq.; H. Lillies, Esq.; J. F. Matthews, Esq.; J. Mudge, Esq., Jun.; C. A. Nankivell, M.B.; S. R. Philipps, M.D.; G. Pyeroff, Esq.; E. M. R. Rendle, Esq.; G. T. Rolston, Esq., Jun.; T. Sanctuary, M.D.; G. J. S. Saunders, M.D.; E. Sharp, Esq.; F. L. Stephenson, M.B.; W. P. Swain, Esq.; G. Thom, Esq.; H. Ubsdell, Esq.; W. Wearne, Esq.; E. L. West, Esq.; C. Whipple, Esq.; W. Whitworth, Esq.; J. Woodman, M.D.

*Hon. Secs. for Cornwall.*—R. S. Hudson, M.D., Redruth.

*For North Devon.*—W. J. Square, F.R.C.S., 22, Portland Square, Plymouth.

*For South Devon.*—H. Davy, M.D., Southernhay, Exeter.

*Returns.*—Cornwall: I., 6; II., 2; III., 4; IV., 2. Total, 14.

*Returns.*—North Devon: 0.

*Returns.*—South Devon: I., 5; II., 4; III., 7. Total, 16.

Total returns from the Branch, 30.

**Southern Branch.**

**DORSETSHIRE.**—*Committee.*—W. G. Bacot, M.D.; Christopher Childs, M.B.; W. E. Good, Esq.; W. A. E. Hay, Esq.; B. Jumeaux, Esq.; J. Comyns Leach, M.D.; A. G. S. Mahomed, Esq.; C. J. Marsh, Esq.; P. W. G. Nunn, Esq.; H. R. Sherrard, Esq.; Hatton Smyth, M.D.; W. H. Williams, M.D.

*Hon. Sec.*—C. H. W. Parkinson, Esq., Wimborne Minster.

*Returns.*—I., 8; II., 4; III., 4; IV., 2. Total, 18.

**EAST HANTS.**—*Committee.*—None appointed.

*Hon. Sec.*—T. C. Langdon, F.R.C.S., Northgate House, Winchester.

*Returns.*—0.

**SOUTH HANTS.**—*Committee.*—H. R. L. Veale, M.D., Deputy Surgeon General, A.M.D.

*Hon. Sec.*—Theoph. W. Trend, M.D., 6, Anglesea Place, Southampton.

*Returns.*—I., 4; II., 2; III., 2. Total, 8.

**ISLE OF WIGHT.**—*Committee.*—The Members of the District.

*Hon. Sec.*—W. E. Green, Esq., Belgrave House, Sandown, Isle of Wight.

*Returns.*—I., 10; II., 6; III., 9; IV., 2; V., 2. Total, 29.

WILTSHIRE.—*Committee*.—None yet appointed.

*Hon. Sec.*—H. J. Manning, Esq., Laverstock House, Salisbury.

*Returns*.—III., 1. Total, 1.

Total returns from the Branch, 56.

### Staffordshire Branch.

*Chairman*.—J. T. Arlidge, M.D.

*Committee*.—J. G. Cleudinnen, Esq.; J. Cooke, M.D.; F. G. Gray, Esq.; D. H. Monekton, M.D.; G. A. Phillips, Esq.; G. G. Sharp, Esq.; J. Y. Totherick, M.D.; J. H. Tylecote, M.D.

*Hon. Gen. Sec.*—Vineut Jackson, Esq., Wolverhampton.

*Hon. District Secs.*—East Staffs.: W. G. Lowe, M.D., Burton. Mid-Staffs.: G. Reid, M.B., Stafford. North Staffs.: A. M. McAldowie, M.D., Stoke. West Staffs.: H. Malet, M.D., Wolverhampton.

*Returns*.—I., 19; II., 8; III., 12; IV., 9. Total, 48.

Total returns from the Branch, 48.

### Thames Valley Branch.

*President*.—J. Langdon Down, M.D.

*Committee and Hon. District Secs.*—C. C. Gibbs, M.D., Surbiton; A. R. Graham, M.B., Weybridge; E. H. Hare, Esq., Kew; N. H. K. Kane, M.D., Kingston Hill; H. H. Murphy, M.D., Twickenham; F. J. Wadd, M.B., Richmond.

*Hon. Gen. Sec.*—F. P. Atkinson, M.D., Surbiton Road, Kingston.

*Returns*.—I. 5; II., 1; III., 3; IV., 2. Total 11.

Total returns from the Branch, 11.

### West Somerset Branch.

*President*.—G. W. Rigden, Esq.

*Committee*.—H. J. Alford, M.D.; J. Meredith, M.D.; W. L. Winterbotham, M.D.

*Hon. Sec.*—W. M. Kelly, M.D., The Crescent, Taunton.

*Returns*.—I., 4; II., 2. Total, 6.

Total returns from the Branch, 6.

### Worcestershire and Herefordshire Branch.

*Committee*.—Tom Bates, Esq.; H. J. Brown, Esq.; J. R. Buck, Esq.; W. F. MacCarthy, M.B.; T. Pike, M.D.; S. S. Roden, M.D.; J. J. Sarjant, Esq.; H. Swete, M.D.; W. A. S. Walsh, Esq.

*Hon. Sec.*—Geo. W. Crowe, M.D., Shaw Street, Worcester.

*Returns*.—I., 10; II., 2; III., 3; IV., 9; V., 1. Total, 25.

Total returns from the Branch, 25.

### Yorkshire Branch.

*Committee*.—The Council of the Branch.

*Hon. Sec.*—A. Jackson, Esq., Wilkinson Street, Sheffield.

*Returns*.—I., 27; II., 9; III., 28; IV., 9; V., 2. Total, 75.

Total returns from the Branch, 75.

### Returns from Members not attached to any Branch.

J. Aikman, M.D., Guernsey: I., 4; III., 1. E. Drummond, M.D., Romo: I., 1; II., 1; IV., 2. Herbert Parsous, M.R.C.S., Monte Video: III., 1; V., 1. Otherwise received, 79. Total, 90.

Total Returns received during the year from June, 1882—June, 1883:—

I. Acute Pneumonia . . . . .	493
II. Chorea . . . . .	228
III. Acute Rheumatism . . . . .	339
IV. Diphtheria—Clinical . . . . .	144
Etiological . . . . .	115
V. Syphilis . . . . .	25
Total . . . . .	1344

Phthisis returns . . . . . 1078

[NOTE.]—To keep the lists necessary for such an account as this entails great labour, which would be considerably lessened if Hon. Members would kindly write on their cards the name of the Branch to which they belong. Otherwise it is impossible to avoid occasional mistakes.



## MEMORANDA AND CARDS ALREADY ISSUED.

---

### MEMORANDUM ON ACUTE PNEUMONIA,

ESPECIALLY WITH REGARD TO ITS ETIOLOGY AND EPIDEMIC PREVALENCE.

BY OCTAVIUS STURGES, M.D., AND SIDNEY COUPLAND, M.D.

(*On behalf of the Committee.*)

THE object of this inquiry is to collect evidence from those who are best able to afford it, bearing on the natural history of acute pneumonia as observed in this country; and especially its etiology. Upon this latter question opinion is much divided, and while in other countries valuable material has been collected respecting it, little has been done in our own. It is now desired that an impartial investigation should be made upon the disease, in the hope that information of great value may be elicited. Such an investigation might reasonably be expected to be of service in the promotion of particular measures of prophylaxis, and probably also in the establishment of a rational therapeutics in this disease.

At the present day, two views are commonly held, concerning the etiology of an attack of primary acute lobar pneumonia in a previously healthy individual. They may be concisely summed up under the terms: 1. Exposure; 2. Infection. The first view is that generally accepted; the question is, What grounds exist in favour of the second? That this latter form of pneumonia does exist, possibly to a far greater extent than is admitted, seems likely, not only from the records that appear from time to time upon "epidemic" and upon "contagious" pneumonia, but also from the well-attested facts of the ordinary course of the disease. All clinical observers are agreed that the fever characterising many of the best marked cases of acute pneumonia does not run parallel with the physical signs of the pulmonary inflammation; that it does not, in other words, coincide with the latter in degree or in duration. For instance, high fever usually accompanies a small tract of inflammation, when this is seated at the apex of the lung instead of at the base; and again, it not uncommonly happens in an ordinary case of basic pneumonia that the fever subsides rapidly (by crisis), some days before the local signs indicate a corresponding improvement in the damaged organ. These are but two examples out of several which might be quoted, as affording *prima facie* support to the view that in the disease we call "pneumonia" there is something over and above the mere condition of an inflamed lung; some influence, call it septic, or what not, which, attacking the whole organism, has its local and manifest expression in pulmonary inflammation. Have we, in a word, in the inflamed lung, a condition related to some underlying influence (at present unknown) in a manner analogous to the bowel affection characterising typhoid fever; or, to the cutaneous inflammation of facial erysipelas; or, on the other hand, is pneumonia simply a local disease, solely due to "exposure," like catarrhal affections?

We have here, however, not to deal with speculations, but only to ask for facts. The facts supplied may go far to show that an "epidemic" of pneumonia means nothing more than a great prevalence of the disease due to atmospheric conditions, to which the term "epidemic" is no more applicable than it is to bronchitis, when that happens to be prevalent. The Collective Investigation Committee invite the profession to aid them in determining a question, the solution of which will materially further the progress of scientific and practical medicine.

The main points to which attention is directed are given in the accompanying schedule. They do not involve detailed statements, and most of them can be answered by a mere affirmative or negative, or by the erasure of certain words. A few brief explanations will suffice to show the purport of the questions.

The answers to the questions concerning *occupation* and *habits* will point to the existence or not of any factors peculiar to the individual, which may operate in rendering him susceptible to the disease. From them it will be learnt whether his life, passed in the counting-house, factory, or workshop, or in the farm or mine, be sedentary or active; whether the occupation, in short, be one likely to expose him to unsanitary or miasmatic influences, to changes of temperature, to great physical fatigue or mental effort, or to other conditions whereby his general health may have suffered, or his liability to "take cold" be enhanced; while, as to habits, it may be expected that some light may be thrown upon the extent to which impoverished diet, and especially *alcoholic intemperance*, conduces to the determination of pneumonia.

Then follow a series of questions specially intended to elicit facts bearing upon the existence of epidemics of pneumonia, and the conditions under which they arise. The *locality* and *situation* of the patient's dwelling, whether this be in an elevated position, isolated and exposed, or sheltered in a valley, or buried among trees, or in the heart of a thickly-peopled town, together with the nature of the soil on which it stands.

It should be explained that, under the next heading, *atmospheric conditions* prevailing at the time of the attack or epidemic, it is only intended to ask for such general statements as "dry," "damp," "wet," "cold," "hot," "changeable," and the prevailing wind—such as come naturally under the head of "weather"—without any detailed "meteorological" data being required; as these could be supplied, when necessary, by reference to the records of the Meteorological Office.

The next query requires a single word in answer from the practitioner. Are there *other cases of pneumonia* in the patient's house or in the surrounding district? If there be other cases, and an outbreak of pneumonia be generally prevalent, the observer's returns upon *each* of the cases that come under his notice will afford the chief evidence of the presence of "epidemic pneumonia," so far as his practice goes. To make the information complete, it is to be hoped that, whenever pneumonia is unduly prevalent in a district, every practitioner concerned will take part in this inquiry. In this way the Committee would be placed in possession of a mass of facts of the greatest value accumulated by independent observers.

It is also of great importance to learn whether, at the time of the prevalence of pneumonia, there be concurrently an undue prevalence of the *specific fevers*, e.g., typhoid, scarlatina, diphtheria, erysipelas, etc., as it may happen that conditions liable to produce such diseases in some individuals may favour pneumonia in others. It must be understood that examples of pneumonia occurring as a complication in the course of a specific fever are not required. Where, however, as sometimes happens, pneumonia occurs in the *initial* stage of a specific fever—notably typhoid—such a case should be recorded in this inquiry. In like manner, information is asked for as to any concurrent undue prevalence of those ill-defined mild febrile conditions, to which the terms *febricula* and *catarrhal fever* are applied, with the view to ascertain whether they also arise under conditions existing at the time when pneumonia prevails.

The next question applies to a different branch of the subject. In asking for a return of the concurrent prevalence of *bronchial catarrh*, it is intended to ascertain how far an "epidemic" of pneumonia may be explained by the existence at the time of meteorological rather than "septic" conditions. If, for instance, the returns show that pneumonia and bronchial catarrh are both unduly prevalent in a particular district, where there is no reason for suspecting any unsanitary influences, but at a time when cold and damp weather is in the ascendant, then surely it may fairly be concluded that the pulmonary and bronchial disease have in this instance the same non-specific etiology; whereas, on the other hand, if pneumonia largely prevail, and catarrhal affections be at a minimum, there will be ground for suspecting that meteorological variations were not alone, if at all, to be assigned as the cause of the pneumonia.

Next on the list comes the important subject of *sanitary conditions*, which, if carefully inquired into, may throw much light upon many an "epidemic" of pneumonia. It may be discovered, for instance, that, when several members of a household have been in succession struck down by the disease (not at the time generally prevalent in the district), the house itself is in an insanitary state, and that its inmates have been

poisoned by sewer-gas or other noxious effluvia. Or, again, the practitioner, meeting with an unusually large number of cases in his district, may find that the drainage is generally defective. If a reasonable doubt exist on these matters, the word "uncertain" will sufficiently express it.

The remaining queries refer solely to the *family* and *personal history* of the patient and the leading features of his attack. From the answers furnished, it will be possible to learn whether there be any uniformity in the extent and seat of the pulmonary inflammation, in the course of the concomitant fever, in the rate of mortality, etc.

Lastly, space is left for any additional remarks from the observer which appear to him desirable; but there is no need for him to go beyond the few points named, as answers to them will furnish all the information required; and he will find that these replies can be made without the expenditure of much time. Facts alone are asked for, and those neither numerous nor abstruse; but they should be plainly and simply stated, unbiassed by views or opinions; and so presented, they will be collated and analysed, with a view to the solution of one of the many vexed questions in medicine.

## No. I.

**ACUTE PNEUMONIA.**—*With regard to (1) its epidemic prevalence, (2) its communicability, (3) its association with other prevalent diseases and with defective sanitary conditions, (4) its symptoms, duration, and result, (5) its treatment.*

Observer's Name .....  
Address .....  
Date of last obs. ....  
(Reply where possible by crasing words on card.)  
Initials of patient. M. or F. Age.  
Married. Single. Widowed.  
Occupation.  
Temperate. Intemperate. Total Abstainer.  
Food—sufficient, insufficient.  
Place of residence.  
Locality—high, low, damp, dry, exposed, confined.  
Prevailing wind at onset of attack.  
Atmospheric condition—Dry, damp, wet, cold, hot, mild, changeable, sun or clouds.

Sanitary condition of house—Good, bad, indifferent.  
" " district—Good, bad, indifferent.

Family history of lung disease.

Previous illnesses of patient, with dates.

Attack preceded by rigors. Date.

Premonitory symptoms.

Date of onset of attack.

Part of lungs affected. R. Base, apex.

L. Base, apex.

Expectoration—Blood, rusty, white, none.

Fever—Severe, moderate, mild.

Highest range of temperature.

Duration of fever.

Termination of fever—Sudden, by gradual subsidence.

Duration of physical signs.

Result.

Remarks on any special feature of case.

Sequelæ.

Plan of Treatment.

How long has patient been under care of observer?

(This card as soon as filled up to be returned to Secretary of the Local Sub-Committee.)

	In same House.	In District.
Number of cases of pneumonia ..		
" any kind of fever* ..		
" catarrhal fever ..		
" tonsillitis ..		
" herpes ..		
" bronchial catarrh ..		
" erysipelas ..		

State number of each disease under care of observer.

\* Nature of prevalent fever.

N.B.—Information and assistance given to the Committee in this and all other investigations will be duly acknowledged.

## MEMORANDUM ON CHOREA.

By STEPHEN MACKENZIE, M.D.

(On behalf of the Committee.)

THE object of this investigation is to gather from a number of different observers their experience as to the conditions that predispose to, or excite chorea. The observers are also asked to note the frequency with which certain phenomena occur, and to mention the common ailments to which sufferers from chorea are especially liable, in



order to ascertain its clinical alliances. Though our knowledge of chorea has, in late years, made some advances, it is generally felt that accurate information as to the antecedents and family history of patients suffering from chorea is greatly wanted. Those engaged in family practice have unrivalled opportunities of collecting evidence on these points, which may greatly advance our knowledge of the disease. The family medical attendant will often have known the patient from birth, will be acquainted with the parents and often with collateral relatives. He will thus be peculiarly favourably placed for observing the constitutional peculiarities of the patient and his inherited tendencies, and for estimating the importance attaching to the circumstances supposed to originate an attack of chorea. The patient remaining under his care subsequent to the illness, the observer will be enabled to note important after-changes.

The questions are arranged in such a way as, first, by means of initials, to identify the patient, to record the sex and age, the social class to which the patient belongs, and place of residence; next, to ascertain individual peculiarities such as nutrition, vigour, and growth; the previous mental condition, as to excitability, stupidity, quickness, slowness, etc.; in the female, whether menstruation be regular, or whether the patient be pregnant; and the conditions as regards sufficiency of food.

Information is particularly desired as to immediately or remotely antecedent illnesses; and, in order to ascertain their influence, the observer is asked to note their date or duration. As regards rheumatism, which, as most agree, bears some relationship to chorea, the observer is asked to state whether the antecedent rheumatism was well pronounced, with distinct joint-affection; or whether, as often happens in children, only vague pains and sometimes slight feverishness had been present. Attention is directed to scarlet fever and anæmia as predisposing to chorea; and in these connections, as in the preceding, it is important to note most carefully, under the proper head, the known condition of the heart prior to the attack of chorea recorded. Space is left for other diseases to be noted, whether they have or have not a known bearing on the production of chorea. The supposed exciting cause, and the interval between it and the attack, should be noted, in order that its importance may be estimated under this head. Attention is directed to overwork, mental and bodily; shock and fright, or any other cause. This closes one chapter of the inquiry, and brings us to the actual attack.

It should be stated from how many previous attacks of chorea the patient has suffered, in order that, in the analysis proposed to be made of the answers, cases of first attacks may be considered separately from those where several have occurred. It is then asked whether the attack be severe, moderate, or mild. No attempt is made to define these grades: ordinary experience will enable any case to be placed in one of the three categories. It is also asked whether the temperature were normal, sub-normal, or febrile. The date of onset is to be noted, and, at the conclusion, the duration and result of the attack.

Next, we come to the condition of the heart. For convenience, the record of the state of the heart before the attack, *when known*, is placed here. When no previous examination of the heart has ever been made, it should be recorded *not known*; when the heart has been examined previous to the attack, it should be recorded "normal," when such is the case; or, when diseased, the observed abnormality, as mitral systolic, aortic systolic, aortic diastolic, etc.; and, when practicable, any displacement of the apex-beat. Thus, to fill in an ideal card, under condition of heart: "*Before attack*.—Mitral systolic, apex-beat to left of nipple, heaving." This would enable anyone to recognise, equally with the observer, that mitral regurgitation, with hypertrophy of the left ventricle, existed previous to the attack of chorea recorded on the card. The observer is next asked to record in the same way the condition of the heart noticed during the attack; and, at the conclusion of the case, whether any peculiarities noticed previously to, or during, the attack persisted or disappeared. Irregularity of the heart, and other alterations, besides murmurs, should be carefully noted.

Information is asked as to the occurrence of rheumatism during or after an attack of chorea, and, when so occurring, as to its severity, as indicated by many or few joints being affected. Space is left for other complications and sequelæ, as paralysis, insanity, etc., being recorded. Attention is directed to the occurrence of "subcutaneous nodules," which are sometimes met with in connection with rheumatism and chorea, and concerning which information is needed. These nodules are usually about the size of a split-pea, though they vary from the size of a pin's head to that of

an almond. They are strictly subcutaneous; the skin over them is not reddened; they are painless. The back of the elbow, the malleoli, and the margins of the patella, are the most common situations of such nodules, but they have been met with over the vertebral spines, the spine of the scapula, the extensor tendons of the foot and hand, the scalp, etc.

Observers are asked to note the common ailments to which the patient is liable, in order to ascertain the clinical associations of chorea. It is desired, also, to obtain information as to the occurrence of nervous diseases and rheumatism in members of the patient's family. This can be succinctly given. Thus, under the first head, in an ideal case it might have to be recorded: Father, insane; paternal aunt suffered from chorea in childhood. Under the head of "Family History of Rheumatism:" Mother had rheumatic fever; one brother, crippled from rheumatism.

A few notes on "Treatment" are desired, when a simple or consistent plan has been employed. Principles, and not details, of treatment should be noted; as, for instance, "arsenic," or "iron," or "sulphate of zinc," or "expectant;" or, "at first arsenic, later quinine."

Lastly, space is left for "Remarks on any special feature of the case." The nature of eruptions, such as various forms of erythema, etc., when occurring, should be recorded, and any other points of interest. The main object of the inquiry will, however, be served by simply answering the questions. When fatal, the mode of death should be noted, and when a *post-mortem* examination has been made, the appearances observed should be recorded.

It may seem that the questions are both numerous and intricate; but it will, it is hoped, be found that the questions run on in a connected manner, and very many require only a stroke of the pen to constitute an answer. When practicable, a positive and negative answer are printed, and all that is required is to strike out the words which do not correctly describe the condition.

## No. II.

CHOREA: (1) *its relation to Rheumatism and other diseases*, (2) *the frequency with which certain accompaniments occur*, (3) *the common ailments to which those who suffer from it are liable*, (4) *the predisposing conditions and exciting causes*, (5) *its treatment*.

Observer's Name .....

Address .....

Date of last obs. ....

(Reply where possible by erasing words on card.)

Initials of patient. M. or F. Age.

Upper, middle, or lower class.

Place of residence.

Locality—high, low, damp, dry, exposed, confined.

Stout, moderate, thin; strong, moderate, weak; dark, fair.

Previous mental condition.

Growth—rapid, moderate, slow.

Menstruation—regular, irregular. Pregnant.

Food—sufficient, insufficient.

Antecedent Illnesses.

Rheumatism. With distinct joint-affection.

Date.

With fever. Date.

With vague pains. Date.

Scarlet fever. Date.

Anæmia. Duration.

Other diseases. Date.

Supposed Exciting Cause of Present Attack.

Overwork—mental, bodily.

Shock, fright.

Other causes.

Interval between exciting cause and attack.  
How many previous attacks.

Present Attack—severe, moderate, mild.

Date of onset. Duration.

Temperature—normal, sub-normal, febrile.

Condition of Heart.

Before attack.

During attack.

After attack.

Rheumatism—Acute, subacute, chronic.

During attack. Many joints. Few joints.

After attack. Many joints. Few joints.

Subcutaneous nodules—present, absent.

Other Complications.

To what common ailments is patient specially liable?

Family history of nervous diseases.—In what members?

Family history of rheumatism.—In what members?

Remarks on any special feature of case.

Sequelæ.

Plan of treatment.

How long has patient been under care of observer?

(This card as soon as filled up to be returned to Secretary of the Local Sub-Committee.)

N.B.—Information and assistance given to the Committee in this and all other investigations will be duly acknowledged.



## MEMORANDUM ON ACUTE RHEUMATISM.

By J. F. GOODHART, M.D., AND THOMAS FARLOW, M.D.

*(On behalf of the Committee.)*

THE common diseases are those which repay study the best; and, in a general survey of systematic medicine, it is wonderful to note how many of what are regarded as fundamental facts in such diseases rest on an insecure basis of observation and experience. There is no country in Europe which affords better opportunities for studying acute rheumatism than Great Britain; but we are still ignorant as to its etiology. We have no accurate data in regard to the influence of food; and, beyond the curious but well-established observation of Dr. Balthazar Foster, on the appearance of a quasi-rheumatic affection of joints, etc., under the administration of large doses of lactic acid, physiological chemistry has, as yet, given us no help. Apart altogether from chemical questions and speculative views as to its bacterial origin, how little we know about the immediate antecedents or determining causes of acute rheumatism! We are in the habit of accepting the conventional lay explanation, that the patient was exposed to cold whilst he was sweating, that thereupon he developed rheumatism; and over and above this we have nothing to say, except that "certain people are more liable to such illnesses than others." But we are bound to take note of cases occurring occasionally in hospital, where patients in bed, suffering or convalescing from some other complaint, develop undoubted rheumatic fever, and where it seems difficult to suppose that the factor of exposure has played any part. The subject of the relation of chorea to rheumatism is to be separately discussed in another inquiry; but, by way of illustration, we may here refer to cases which first come under notice as typical chorea, and then develop unquestionable acute rheumatism. And this leads us to remark that severe nervous shock, induced by accident and other causes, appears now and then to be an immediate antecedent of acute rheumatism. Without attempting any explanation of the connection, it is most important to "keep our minds open" to the nervous relationships or antecedents of the disease, because some of the clinical features of the hyperpyrexial attacks point strongly towards a nervous origin; and, further, the association of some forms of joint-affection with certain diseases of the spinal cord is also suggestive. Amongst other antecedents of rheumatism concerning which careful records are needed, is scarlatina. It is a common enough fallacy to call every affection of a joint which suppurates either strumous or pyæmic, and one which does not suppurate rheumatic; and again and again we are baffled by being unable to give a true definition of rheumatism. But with regard to the joint-affection of scarlatina, it is clear that at least three views may be taken. 1. We may hold that it is one of the manifestations proper to the disease itself—a scarlatinal arthritis. For want of better terms, we may say that the scarlatinal poison affects the joints just as it may affect other parts. According to this view, it would be a mistake to identify the scarlatinal joint-affection with acute rheumatism, although in many respects running parallel to it—notably, in the frequent contemporaneous affection of heart and serous membranes. 2. We may consider it an epiphenomenon, depending on some auto-infection, and therefore septicæmic in character. Although it by no means conclusively proves the septicæmic nature of the affection, it is important to bear in mind that occasionally in these cases suppuration in a joint occurs. 3. We may consider the arthritis in question as that of true acute rheumatism super-added to the attack of scarlatina, and quite as independent of what may be called the scarlatinal poison as an intercurrent varicella might be.

There is much to be said for each of these views, and it is possible that they may be respectively true in different cases. Even those who claim that the affection is true acute rheumatism, are willing to concede that it often occurs as a complication rather than as a sequela; and there would be something gained by observations on a number of cases as to the exact period after scarlet fever at which joint-symptoms arise. The rash being generally a definite thing, it would be advisable to reckon from the day of its appearance.

Much attention has of late been directed to tonsillitis in regard to acute



rheumatism. It would appear in some cases to be an initial symptom of the attack, and in others an antecedent with a distinct interval. More details are needed, and ought not to be very difficult to obtain.

With regard to the ordinary features of a primary acute attack when fully established, there is but little information needed; but the slight later attacks will well repay study, and this is especially the case when the disease is uncomplicated by heart-affection, the results special to which need elimination. The rheumatic attacks of children need special study; the sweating may be very slight, the fever of short duration, the joint-trouble insignificant, whilst, as is well known, the heart-affection is considerable.

In regard to the arthritis of acute rheumatism, it is generally postulated that the subsidence is complete, although there is proneness to relapse. But there are some alleged cases of suppuration having occurred as a sequel of rheumatic fever, where scarlatina was out of the question. Of such very rare occurrences it would be valuable to get some further well-recorded examples, in which the early part of the attack is perfectly typical. The same may be said in regard to cases of fibrous thickening around and ankyloses of joints, after acute rheumatic attacks, where gonorrhoea can be excluded.

In children who are the subjects of heart-disease presumably rheumatic, and sometimes in adults also, subcutaneous nodules are to be found in the neighbourhood of both large and small joints, and on the hairy scalp and the forehead, varying in size from a pin's head to an almond. They often appear in crops, and as they are generally painless, and the skin over them most commonly unaffected, they need to be searched for; and information is particularly required concerning their relation to active disease of either heart or joints.

There is ample scope for observation in regard to the skin-eruptious associated with acute rheumatism. It is now well recognised that polymorphous erythema is often an intercurrent phenomenon in rheumatic fever, though the cases in which it occurs are sometimes ill-defined in regard to arthritis. Records of cases are needed in which urticaria and purpura have been observed during the course of, before, or after an undoubted acute attack. A case often referred to by Sir William Jenner may here be quoted in illustration. A patient was brought to hospital with signs of acute pericarditis, but no joint-trouble. Urticaria appeared, which convinced Sir William Jenner that the case was truly rheumatic, and this was soon confirmed by subsequent characteristic appearances about the joints, and the further progress of the case.

The number of questions which gather around the subject of rheumatic heart-disease is legion; but there is at least one which is eminently practical, and the solution of which can only come from the records of family practice—viz., in what proportion of cases in which there is an unquestionable murmur present during the attack does the murmur entirely pass away, the heart remaining sound so far as physical examination can establish? It has been too lightly assumed that such transitory murmurs are hæmic, so called; but it would be better for clinical purposes to eliminate the term altogether, and simply record auscultatory observations and notes on cardiac dulness and impulse in regard to the question of dilatation.

Leaving now the history of the attack, we come to some simple questions in regard to the patient which, if answered on a sufficiently large scale, may assist in building up a definition of what is meant by the rheumatic diathesis. These relate to the common ailments to which the patient is specially subject, and the previous illnesses for which he has needed the doctor's care.

On the subject of the treatment of acute rheumatism, it may be truly said that in no disease are more careful notes necessary as to the entire duration of the illness. Furthermore, it is of the greatest practical importance to get definite statements as to the length of time in the convalescent period during which special drugs are administered, with a view to obtaining collective experience as to the avoidance of relapse.

Appended is a copy of the form which has been adopted for the collective investigation of this subject. And it may here be emphatically stated that the Committee desire to give the fullest recognition to individual observations bearing not only on the questions here indicated, but on others cognate to them.

## No. III.

ACUTE RHEUMATISM: with regard to (1) its antecedents, (2) the frequency with which certain symptoms occur, (3) the common ailments to which those who suffer from it are liable, (4) the climatic conditions with which it is associated, (5) its treatment.

Observer's Name .....  
Address .....  
Date of last obs. ....

(Reply where possible by erasing words on card.)

Initials of patient. M. or F. Age.  
Married. Single. Widowed.  
Occupation.  
Temperate. Intemperate. Total abstainer.  
Food—sufficient, insufficient.  
Place of residence.  
Locality—high, low, dry, damp, exposed, confined.

Atmospheric condition—dry, damp, wet, cold, hot, mild, changeable, sun, clouds.

Prevailing wind at onset of attack.

Recent Antecedents.

Scarlet fever. Date  
Tonsillitis. Date  
Pharyngitis. Date  
Other diseases. Date  
Exposure to wet, to cold. Date  
Over-fatigue, sudden, prolonged. Date  
Shock. Date

Date of Onset.

Attack severe, moderate, mild.

Duration of fever days, of pain days.

Whole duration of attack.

Sweating—slight, considerable.

Extent of Joint Affection.

Many joints. Few joints.

Migratory. Fixed.

Result—recovery complete, partial, death.

Persistence of disease in one or joints.

Suppuration in one or joints.

Anchylous of one or joints.

Heart Affection. Pericarditis. Murmur.

Before present attack

During.....

After .....

Position of apex beat

Skin Eruptions. Nature. Date.

Before present attack

During .....

After .....

Subcutaneous nodules—present, absent.

How many previous attacks.

Age at first attack.

To what common ailments is patient specially subject?

For what other diseases has observer attended patient?

Remarks on any special feature of case.

Sequelæ.

Plan of Treatment.

How long has patient been under care of observer?

(This card as soon as filled up to be returned to Secretary of the Local Sub-Committee.)

## MEMORANDUM ON DIPHTHERIA.

By SHIRLEY F. MURPHY.

(On behalf of the Committee.)

THE intention of this inquiry is to obtain information as to—

1. The conditions which give rise to diphtheria, and the means by which it is communicated.

2. The relation of diphtheria to other diseases.

3. The behaviour of the disease in the individual attacked.

Any one studying the etiology of diphtheria, or indeed any of the infectious diseases, must endeavour to learn accurately the conditions associated with the existence of the disease amongst those attacked, and compare them with the conditions associated with those who escape. By means of this comparison, it becomes possible to eliminate those conditions which are common to both groups of individuals—viz., those who are attacked, and those who escape; perhaps to identify some condition as specially co-existent with the disease, and, finally, to accumulate sufficient evidence to enable it to be regarded, in its relation to the disease, as cause is related to effect.

An inquiry such as this must necessarily include a study of all the conditions of life, and must be undertaken for many districts, those where the disease under consideration is peculiar for its absence being not less deserving of careful study than others in which it has been exceptionally prevalent. Rural districts, more than others, best promise to reward the investigator for his labours: for, in such

regions, the habits and movements of every individual are far better known, and the probabilities of exposure to any existing contagion can be better estimated, than in populous cities.

When it is found that a disease is constantly more prevalent in any one locality than in neighbouring districts, these should be carefully compared. The geology of all should be studied; the altitude; the condition of the soil; the opportunity for exposure to different winds; the drainage and mode of dealing with refuse; the water-supply; the food, clothing, and occupations of the inhabitants; the relative proportions of old and young persons; the extent to which marriages of consanguinity occur; all diseases to which the inhabitants are subject, as well as the ethnological peculiarities of the latter; the liability of the inhabitants to exposure to diseases of animals, as well as the relative opportunities which exist for the introduction of disease from other places.

Following the same lines, it should be noted whether the disease is habitually more prevalent in one part of a town or village than another; and if any local condition coexists with this special prevalence in the one part compared with the other.

Again, when an individual household only is concerned, where it may be possible at once to eliminate some of the larger conditions which must be common to the rest of the town or village, it becomes necessary to take cognisance of everything relating to the house itself and its occupants.

Observation should be directed to the possibility of the person attacked being exposed to some known source of infection.

The incubation period of the disease should be borne in mind, and an inquiry made into the movements of the patient at the time which is indicated as that at which the infection must have been received, such as would expose him to infected persons or infected things by visiting or otherwise.

Failing evidence of infection being received from some source which can be traced, the degree of probability should be noted (a) whether infection may have been received from some unknown person suffering from disease or from some infected article; (b) the extent to which such possibility can be excluded.

The opportunity for excluding the ordinary means of conveying infection will be found more often in country places, where every individual and his condition of health is known, than in more populous places. It is in the latter class of case that an examination of the house in which the patient resides and its contents becomes of more value, and especially in those cases in which the patient has not left the house for some time before his illness began.

When two or more members of a family are simultaneously attacked, the probability of infection being received from a common source is considerable, and an inquiry into the conditions common to both, and which differ from conditions relating to those who escape, should be made, regard being had to the question whether all are equally susceptible and equally exposed to suspected infection.

In all such cases, an examination of the house should be carefully conducted, and the condition recorded. It should be particularly noted whether the house has any relation with neighbouring houses, either in its drainage or its water-supply, and the existence or absence of cases of illness in these ascertained.

If other cases are found to be occurring in the neighbourhood, the medical officer of health should be at once informed of the outbreak, and should be supplied with information of the locality of the house, the number of persons in each household, the number of persons attacked, and the date of the beginning of illness in each, together with the names of the tradesmen supplying the various articles of food. If any number of persons, especially if living in different houses, be simultaneously attacked by any infectious disease, there is a probability that the outbreak is due to an infected food-supply. Thus, infected milk or infected water will simultaneously produce certain infectious diseases in a proportion of those persons who have taken it.

Care must be taken to distinguish between the first case of such a disease introduced into a household, and secondary cases arising from the first by means of direct contagion. No conclusion must be come to from positive evidence alone, without due regard being had for any negative evidence which may be forthcoming. It must not be assumed because a majority, or even a very large majority, of those attacked with disease have their food-supply from a particular source, that the food is responsible for the disease; nor will it do to assume that, because some sanitary defect is found in a large majority of the houses attacked, this defect is the occasion of the illness.



It is, first of all, necessary to ascertain the total number of houses in a district with the food-supply or defect, as well as the total number without, and then to learn whether there is any special incidence of the disease upon the former. For this purpose, inquiry should be made at all the houses in a district.

It will be evident that an inquiry of this character must be mainly conducted by some investigator holding an official position, such as medical officer of health. While he, however, is engaged in ascertaining facts concerning the district generally, and especially relating to those houses which are free from illness, he should be receiving information concerning the households in which the disease has occurred, from those investigators who are themselves engaged in the treatment of the affection.

It cannot be too strongly urged that the investigation should be conducted on a well-understood and common line, in order that the observations of each individual may add to the evidence which must be forthcoming to enable a definite opinion to be eventually formed as to the cause of the outbreak. The cards which are issued by the Committee will, it is hoped, serve for this purpose; and it is recommended that the medical officer of health should be supplied immediately with a copy of the information sent to the Committee. The cards have, therefore, been drawn up with the object of enabling the investigator to report on conditions relating generally to the district, and especially to the houses in which the disease has occurred; while a second card has, on the one side, reference to the members of the household attacked, and, on the other, to the patient, and the illness from which he is suffering.

It will be seen that the intention of the inquiry is to enable the Committee to ascertain the conditions in any community special to those who suffer from diphtheria; and they hope that, by a careful analysis of observations made concerning individuals, households, villages, towns, or rural districts, evidence of value will be forthcoming.

In investigating the cause of diphtheria, regard must be had to the fact that the symptoms of this disease vary in different individuals very considerably; that in association with those cases in which a false membrane is found are other cases presenting no other symptom than that of a trifling sore-throat; nor must it be forgotten that occasionally paralysis is the first symptom which gives rise to the suspicion that diphtheria has visited a household. Cases of sore-throat or of paralysis must be carefully inquired for as possibly antecedent to one of well-marked diphtheria, and especially when, for the purpose of identifying a possible cause, the date of introduction of the disease into the household is a matter of importance.

In connection with this may be urged the importance of losing no opportunity for ascertaining as exactly as possible the incubation period of diphtheria; it must not be forgotten that those cases are of the greatest value in which the receiver of infection is exposed to it but for a limited time, and all other sources of infection can be eliminated.

Another point especially deserving the attention of investigators is that of the possible cultivation of a simple sore-throat into well-marked diphtheria by means of transmission through a number of persons living under circumstances favourable to its development.

Diphtheria is a disease which is believed to occur more frequently in rural districts, where the opportunity for communication from person to person is less than in thickly populated towns. This point is especially deserving of observation, and no efforts should be spared to verify or disprove the assertion. Again, it is believed to be more constantly prevalent in some rural districts than in others; and, if this be found to be the fact, there is some promise that a special local condition may be found to be related to the cause of the disease.

The question of food-supply is a very important one. That diphtheria can be communicated by milk there is considerable evidence to show; care should therefore be taken to note the milk supply in all cases, so that evidence may be accumulated on this point. If a number of persons who are attacked with diphtheria are known to be receiving milk from one source, this is reason enough for ascertaining whether the suspicion is well founded, and the medical officer of health should be communicated with; but, as has already been pointed out, no conclusion must be arrived at until it is ascertained that, in the absence of any other possible cause, the incidence of the disease upon the suspected milk, as compared with the incidence of disease upon other milk-supplies in the same district, makes this a practical certainty.

Whenever milk comes to be suspected, the means by which it has become infected are of the utmost importance. Perhaps it will be found that some person about the

farm, or concerned in the distribution of milk, may, himself or his family, be attacked with diphtheria. It must not be hastily assumed that this individual is necessarily the cause of the outbreak, but care must be taken to ascertain whether he has not himself contracted the disease from the same source as the other sufferers. On this point, the date of attack will have an important bearing.

The possibility that the disease may be communicated to the milk from some animal must be borne in mind; even if no ailment can be found among the cows, pains should be taken to trace back the infected supply by the process of differentiation to an individual cow, if this be possible. That the dilution of infected milk by wholesome milk obtained elsewhere might lessen the effects upon the drinkers, ought to be recollected, and the results of drinking unmixed suspected milk compared with the results of drinking a mixture of the suspected milk with some proportion of other milk known to be wholesome; the wholesomeness of the latter should, of course, be proved if possible by the fact of its distribution elsewhere without harm resulting.

The other food-supplies must not be forgotten, and the least suspicion should lead to any clue being followed to its end. The possibility must also be recollected of food becoming infected at any period of its existence up to the time of its consumption.

It may be well also to recollect that the existence of diphtheria has been attributed by different observers to the growth of fungi in a house, and to infection air-borne from a distance.

We now come to the second branch of the inquiry.

Whether diphtheria is related to other diseases, such as scarlatina, measles, enteric fever, erup, or non-membranous sore throat, should be carefully studied. It should be observed whether persons suffering from any one of these diseases, and developing membranous inflammation, have been previously in any way exposed to infection from the latter, or whether they communicate this affection to others.

Again, the chief features of the disease should be noticed, and recorded on the cards provided for this purpose. It is desirable to ascertain whether diphtheria varies in relation to any peculiarity of the individual attacked, the conditions under which he lives, or the manner in which he has received the infection. That certain families suffer more than others from some infectious diseases, there can be but little doubt; and it has been suggested that the conditions under which the sufferer habitually lives have a bearing on the character of his attack.

There is reason to suspect that the means by which disease is communicated may have some bearing upon the severity of the illness; thus the scarlet fever which has been believed to be due to an infected milk-supply has frequently been of a very mild character. This point in reference to diphtheria is therefore deserving of consideration.

Information is also required as to whether a previous attack of diphtheria confers any protection against a second, or whether there is a further susceptibility to the disease, or again, whether a family tendency to sore throat is accompanied by a special liability to attack from diphtheria.

Finally, with regard to treatment, the Committee will be glad if the fact is noted whether the patient was treated with or without alcohol, and if tracheotomy was performed, whether early in the disease as an aid to local treatment or only after asphyxia threatened life. The result will of course be stated.

These are some of the points which the Committee are desirous the investigators should observe, but many others are sure to present themselves in the course of an inquiry into the behaviour of this disease. Every observation should be carefully and exactly recorded, and pains taken to test and ensure the accuracy of any statement made to the investigator.





## No. IVa.

**DIPHTHERIA: ITS ETIOLOGY:—**(1) *prevailing atmospheric conditions*, (2) *amount of rainfall*, (3) *population*, (4) *geological and physical conformation of district*, (5) *sanitary conditions of district*, (6) *sanitary condition of house*, (7) *source of food supply*, (8) *prevalent diseases of men: of cattle*.

(Reply where possible by erasing words on card. When in doubt refer to accompanying memorandum paper for explanation.)

Observer's Name .....

Address .....

Date of last obs. ....

(Please return this card when filled up to Secretary of Local Sub-Committee.)

Initials of patients resident in house, the condition of which is herein described .....

House—isolated, detached, semi-detached, undetached.

Locality of House—high, low, dry, damp, exposed, confined.

Excrement Removal. House drained by main sewer, cesspool, ash pit, earth closets.

House drains—brick, earthenware. Have they been tested and found water-tight?

„ „ under, outside house.

„ „ ventilated, unventilated. Diameter of ventilating pipe.

„ „ form of trap between house and main sewer.

Is there an opening on house side of trap, so as to form atmospheric disconnection?

Main sewer ventilated, unventilated.

Openings of ventilators at road level, above road level. How far apart?

Sinks, lavatories, bath pipes connected, not connected, directly with main sewer.

Are there any pan closets and containers?

Is soil pipe ventilated, and by what sized pipe?

Cesspool.—Ventilated, unventilated—overflow, no overflow—water-tight, porous.

Water Supply—By pipes from reservoir.

By well, surface or deep.

By running stream.

By rain-water cistern.

Water Supply from Reservoir.

Supply common to large district, few houses, or single house.

Conveyed to houses by pipe, by running stream.

Water Supply from Well.

Depth of well.

Structure—brick, stone, iron tube.

Nature of soil.

Inclination of surface of land from cesspool, drain or soil pipe—towards well, from well.

Cess-pit, drain or soil pipe distant yards from well.

Well—foul, clean.

Water Supply from Running Stream.

Exposed to house drainage higher up.

„ „ cattle sewage or droppings.

Water Supply by Rain-water Cistern.

Open, covered.

Exposed to sewage contamination.

Overflow connected with drains or into ditch.

Cisterns for Drinking Water.

Structure—lead, iron, zinc, slate, wood.

Open, covered; inside, outside house.

Clean, foul.

Connected, not connected with W.C.

Waste pipe connected with drain or open air.

Cistern supplying one or several houses.

Nuisances adjacent to House, Water Supply, or Larder.

Dust bins—clean, foul.

Dung heaps—removed frequently, infrequently.

Farm yard—clean, foul, drained, undrained.

Stables—clean, foul, drained, undrained.

Piggeries—clean, foul, drained, undrained.

Neighbouring houses in process of demolition.

Neighbouring accumulation of rubbish from dust bins for making foundations, levelling land, or other purposes.

Presence of fungus growth or “mould” in sleeping or dwelling rooms.

Other nuisances.

Food Supply.

Name of milkman.

„ butcher.

„ baker.

„ greengrocer.

„ butterman.

Any similar illness in house of either tradesman.

Details concerning the District.

Prevailing atmospheric condition previous to attack—dry, damp, wet, cold, hot, mild, changeable, sun, clouds.

Prevailing wind.

Rainfall of preceding month—excessive, moderate, scanty.

Town, village, country district.

Population—dense, scanty, isolated.

Geological formation—on surface.

„ „ deep.

Altitude.

General conformation—hilly, flat, undulating, woody, bare, marshy, dry.

Situation of house—on hill top, hill side, at foot of hill, in the valley, on the flat, sub-soil wet, subsoil dry, adjacent to standing water, running water, shut in by trees, much herbage, barren.

Prevailing system for the disposal of sewage.

Prevailing water supply.

Prevailing system of refuse removal.

Diseases recently prevalent.

Diseases of cattle recently prevalent.

Other conditions worthy of notice.

## MEMORANDUM ON INHERITED AND ACQUIRED SYPHILIS.

By C. MACNAMARA, F.R.C.S., AND THOMAS BARLOW, M.D.

*(On behalf of the Collective Investigation Committee.)*

THE present inquiry (suggested by the valuable investigations of Mr. C. Palmer, of Great Yarmouth) is concerned with the effects of syphilis on the civil population of this country. An answer to the fundamental question of the extent of prevalence of the disease in different parts of the United Kingdom is, for many reasons, a very difficult thing to arrive at except in general terms. The Committee, in preparing a list of questions, have asked, as the final one, for an opinion as to the relative frequency of the disease in any given district; but they are aware that the answers to this question must necessarily be vague and difficult to summarise statistically. They believe that, if a number of practitioners in different parts of the country will take the pains to fill up a few details of all the cases of syphilis in their respective circles of practice, a definite start will be made, and that in this way, at all events, the relative prevalence of the disease in different parts will not be over-estimated. Mr. Palmer has truly pointed out that a very large amount of acquired syphilis escapes observation and proper medical treatment in the early stage, and that a careful investigation of cases of hereditary syphilis gives evidence of a much larger amount of the acquired disease than would otherwise have been suspected. The Committee, therefore, attach great importance to the form No. Va., which refers to inherited syphilis; and upon this, in the first place, a few explanatory observations are offered.

In this, as in all the other forms issued, the replies are as far as possible to be made by allowing those words to remain which denote the symptoms present in any given case, and crossing out the words which denote symptoms not present in that case. But more space is left for the observer to fill in explanatory detail than has been allowed in the other forms already issued.

The syphilitic children who will be available for the purpose of investigation will be either (A) infants first noted when about six weeks of age, and subsequently kept several months—two years, if possible, under observation; or (B) children who have cut the permanent upper median incisors. The form No. Va. can be used for either of these.

As to the infant of six weeks old, there can be little difficulty in recognising the disease when the signs are definite; but it is certain that many children—marasmic from bad feeding and neglect—are labelled syphilitic, in whom the proof of syphilis is wanting.

With respect to general nutrition, it has been pointed out that occasionally syphilitic infants, so far from being like "little old men," may be very well nourished indeed. The term "earthy colour," which is used in the form, refers, not to the colour of the rash, but to the complexion; it is often most noteworthy after the rash has disappeared, and may, indeed, continue for several months. The symptom of hoarseness is quite as important as that of snuffles, as in syphilitic children it often alters the character of the cry for a long period. With regard to eruptions, a minute description of their character is not needed, but a note as to their situation is of some importance; for it will be conceded that squamous skin-lesions about the mouth and chin, and on the trunk, legs, and soles, are often of more value than those on the nates, where the results of faecal and urinary irritation may closely simulate syphilides.

It has been noted in a goodly number of cases that syphilitic children have undergone vaccination badly. In some, for instance, the conditions after the vaccination have been abnormal. The vaccine soro has been apt to ulcerate, and has been long in healing. It is desirable to note, therefore, in all syphilitic children, the results of vaccination. It may be here pointed out that true vaccino-syphilis—that is, syphilis produced by vaccination—is such a definite thing, that it is difficult to find any excuse for some of the vague general assertions which have been made on the subject. From Mr. Hutchinson's investigations on the subject, it is clear that no case of alleged vaccino-syphilis can be accepted as such, unless there be a distinct interval of



a month to six weeks between the making the vaccination-puncture and the development of a definite hard chancre on the site of the cicatrix, which has been left after the healing of the proper vaccination sore.

It has been noted more than once that vaccination of an infant at three months old has been followed by a true congenital syphilitic rash, but on careful investigation it has been found that the child belonged to an unquestionably syphilitic family; and the study of family groups often establishes cases of delayed specific manifestations. It would be obviously unfair to cite such a case as one of vaccino-syphilis, but it is important that it should be noted.

The symptom, "SWELLING OF LONG BONES NEAR THEIR ENDS," needs a special note, because it is necessary to distinguish it from the common rickety swelling of the epiphyses, with which all are familiar.

A typical case may be given as follows:—A syphilitic child, about the time of the subsidence of the rash, has been noticed to cry a very little when the wrist or elbow, on one or both sides, has been washed, and not to use the said wrist or elbow as much as the corresponding one. The symptoms may be so slight that the medical man's attention is not drawn to it by the mother. Sometimes, however, the droop is so marked as to raise the suspicion of nervous disease, and such cases have been mistaken by very good observers for infantile paralysis. But as there is no wasting, and no alteration of reaction to faradism, the term "pseudo-paralysis" has been properly applied to the condition.

The part is not hot, and only very slightly tender on examination. There may be a very little swelling just above the junction of the epiphysis of the radius with its shaft. But the drooping is a more characteristic symptom than the swelling.

In a week's time a similar description will apply to the corresponding end of the bone of the opposite side, whilst the swelling and partial loss of power are lessening in the part first affected. Within a fortnight or three weeks possibly the ends of all the long bones may be affected more or less; but the affection is found most commonly in the neighbourhood of the wrists, the elbows, the shoulders, and the knees. The amount of swelling may be almost nil, although the powerlessness is definite. Rarely suppuration occurs, which may extend into the joint. Occasionally partial dislocation of the epiphysis from the shaft ensues, with subsequent welding of the epiphysis with some displacement to the shaft, slightly altered from its proper relation. The commonest event of all, so far as can be determined clinically, is for complete recovery to take place spontaneously within about a month. The changes giving rise to these symptoms are chiefly endosteal at the junction of the shaft with the epiphysis. But there is also a varying amount of inflammation of the periosteum or the perichondrium present which gives rise to the slight swelling which may extend up the shaft for several inches. There may be periosteal thickening on the middle of the shafts, but this is rare in the infant period.

ENLARGEMENT OF THE LIVER, although it ought to be noted, because it is often present in congenital syphilis, has but little value as a confirmatory symptom: first, because the liver is proportionally large in infancy, and it is difficult to state the limit of what is actually normal; and, secondly, because other causes besides congenital syphilis lead to its enlargement. With regard to ENLARGEMENT OF THE SPLEEN, the case is different. Dr. Gee's observation, that, in the early stage of infantile syphilis, some enlargement of the spleen occurs in a large number of cases, has been abundantly confirmed. Although, with the subsidence of the other symptoms, this enlargement often disappears, so that on *post-mortem* examination, two or three months after, there may be no trace of it; yet in a few cases it persists, and indeed sometimes increases, so as to be considerable when the other signs have quite vanished. The importance of this sign is greatest when noted early, as, for example, when the child is from two to three months old, for at that period the enlargement of the spleen, due to rickets, can hardly come into question.

The term NATIFORM SKULL, as used by M. Parrot, needs some explanation. If a number of syphilitic infants be carefully watched in regard to the shape of the skull up to the age of twelve months, it will be found that in some of them lenticular swellings on the bone appear nearly symmetrically around the anterior fontanelle, but at a short distance from it; that is to say, one on each frontal, and one on each parietal bone, which may be described as bossed. These swellings are at first tolerably circumscribed, and often measure, in a child nine or ten months old, an inch in diameter. More or less circular at first, they tend to become diffused and massive, and ultimately



organism, giving rise to a more or less thickened skull. It is unquestionable that many of the children presenting such cranial swellings are rickety; it is equally true that these bosses may be found in syphilitic children presenting no signs of rickets. It is very important that the significance of the sign should be worked out.

B. In considering the cases of syphilitic children who first come under observation after the second dentition, it is exceedingly important to have the confirmatory details accurately stated.

With respect to the **PROMINENT FOREHEAD** which is enumerated as one of the signs, it would, of course, be absurd to maintain that every large head belongs to a syphilitic child. Many rickety children have broad square foreheads. But the prominence here referred to is near the middle line, in fact, at and within the frontal eminences. It was insisted upon by Mr. Hutchinson many years ago, and is probably the result of the diffuse extension and organisation of the bossed condition of the frontal bones above mentioned.

Mr. Hutchinson's observations on the characteristic **INTERSTITIAL INFLAMMATION OF THE CORNEA** in congenital syphilis are now well known and generally accepted. It is essential to distinguish between scrofulous ulceration and this characteristic syphilitic disease. The latter is usually symmetrical, and attended by a diffuse ground-glass condition, resulting in general haze and opacity, but throughout wholly free from pustules or ulcers. Very rarely, however, it is attended by peculiar crescentic patches of congestion, the so-called "salmon-tinted patch." It is important to bear in mind the late period at which this form of disease may first appear; as late even as the age of thirty-five years. Also it may be here mentioned that, although the heading of the second division of this form is B., child (after second dentition) age —, adult cases of inherited syphilis may, without violence to the scope of the inquiry, be included under it. The deep affections of the eye have not been referred to in the form, but the Committee will be exceedingly glad to have notes appended as to the presence of *choroiditis disseminata* in one or both eyes, which is almost as valuable a confirmatory detail as the special corneal affection. Notes of any nervous affections which have been observed may also be appended.

With regard to scars round the mouth, it is worth noting that they are of most value when narrow cicatricial lines extend right across the mucous membrane of the lips, especially if there be a radiating series of them. Occasionally careful observation will establish a network of linear cicatrices on the upper lip and round the nostrils as well as at the corners of the mouth and on the lower lip; and when present, this is quite pathognomonic.

The **CHARACTERS OF THE TEETH** are so valuable when present, that it is important to have them carefully noted; the more so that, in spite of Mr. Hutchinson's clear description, they have been much misrepresented. It may be pointed out—1. That only the upper median permanent incisors are characteristic, and sometimes only one of them is typical of the disease; 2. That these teeth are generally a little apart, instead of being in apposition, and are more or less dwarfed; 3. That, in a typical specimen, the width of the cutting edge is narrower than the width of the tooth as it emerges from the gum; 4. That a typical syphilitic tooth presents a single notch, not a serrated margin; and that occasionally, if the notch has not been actually scooped out, there is a little lunula-shaped area, as shown in the left-hand drawing, which, it is easy to see, may readily become a notch; 5. Finally, that, although such teeth, when present, are absolutely pathognomonic, the existence of normal permanent upper median incisors by no means excludes the existence of hereditary syphilis.



The **DEAFNESS** of inherited syphilis is often only slight and temporary; but in many cases it is permanent and almost absolute. It is almost invariably symmetrical, and is, for the most part, unattended by pain or other subjective symptoms.

With regard to the **PERIOSTITIS** of long bones in inherited syphilis, it is worth notice that, as compared with the acquired form, it is often much more extensive, is associated with more hyperplasia of bone, and is very much less painful.

Stature and weight are asked for, because it would appear that occasionally a decided stunting in general growth occurs.

It will be found that the history of infantile syphilitic symptoms of the B group is often imperfect and unsatisfactory.

The headings which follow—viz., present and past evidences of syphilis in the

father and mother—apply, of course, to either A or B. The remaining inquiries need no further remark.

No. V. deals with cases of ACQUIRED SYPHILIS. The most valuable cases are, of course, those coming under observation *ab initio*. Here the first difficulty that arises is as to the nature of the chancre. About some primary sores the observer can have no question whatever; but as to the interpretation of others most practitioners will admit that they have occasionally been at fault, as proved by the sequel. It is proposed, therefore, that a form No. V. be commenced for every case of chancre coming under treatment, and the words "hard," "soft," and "doubtful" will include those which are equivocal in their characters at the time of first observation, as well as those which are definite. The observation for each case should extend, if possible, over two years at least; and it is recommended that it should be sent in, however few be the number of observations made as to the subsequent progress of each case.

The AFFECTIONS OF SKIN AND MUCOUS MEMBRANES, which form the first two categories, are understood to apply exclusively to the early secondary symptoms attacking those tissues. Although, of course, these lesions are generally only superficial, a record of any deep lesion occurring at this period is of great interest. The earliest period at which iritis may occur is also important. The periostitis, about which information is asked in the next line, is the early form, which is sometimes widely distributed, though giving rise to little local swelling, and generally transient. It is very desirable that the distribution and character of the ulcerative lesions which may appear late in the secondary stage should be carefully given. Recent investigations tend to show that gummata may occur much earlier in the progress of syphilis than had been supposed. We have no data as to the earliest period in which they are to be found in the viscera; but in certain cases a temporary (? general) enlargement of both liver and spleen have been observed in the secondary stage. Functional disturbances of the chest or abdomen, however slight in the secondary stage, are worth recording, with dates, under the head of visceral affections; and here also may be inserted a note of the early affection of the testes.

Affections of the nervous system in the early stages of acquired syphilis are probably not uncommon, but much information is needed: for example, on the date of the earliest occurrence of severe headache, mental disturbance, and deafness, all of which may completely pass away. Pathological study of syphilitic brain disease—more especially that depending on specific lesions of the arteries—shows that, although a very wide range of time must be allowed, yet arterial disease may start very early indeed—perhaps even within the first two years, but certainly within the first three years after infection, and sometimes in spite of very thorough early treatment. It would, therefore, be of very great value to get records of hemiplegic attacks and unilateral fits in young adults whose syphilitic history has been carefully followed *ab initio*. The earliest date of occurrence, and the duration of localised paralyses, such as ptosis, paralysis of the sixth nerve, etc., also merit record.

The rigid definition of tertiary symptoms in our present attitude with regard to syphilis is almost impossible. But, for this inquiry, they may be roughly considered as those which appear after a definite interval of health has occurred, and which correspond with lesions for the most part non-symmetrical.

As the present inquiry is to extend over two years only, it may be said that the difficulty of enumerating tertiary symptoms will scarcely arise with those cases which are taken *ab initio*; and, with regard to other cases, if the characters and situation of any lesion be given, it matters little whether the terms secondary or tertiary be employed. The remaining inquiries on treatment, etc., require no comment.

Nov. 22nd, 1882.

## No. V.

**SYPHILIS—ACQUIRED:—**(1) *age of patient*, (2) *primary sore*, (3) *constitutional symptoms*, (4) *duration of symptoms*, (5) *date of development of tertiary symptoms after infection*, (6) *communication to others*, (a) *by direct contagion*, (b) *to offspring*, (7) *treatment*, (8) *prevalence*.

Observer's Name .....  
Address .....  
Date of first obs. ....  
Date of last obs. ....

(When in doubt about purport of any question refer to accompanying Memorandum for explanation.)

(Reply where possible by erasing words. This paper when filled up to be returned to Secretary of Local Sub-Committee.)

Initials of patient or case number.

M. or F. Age.

Married. Single. Widowed.

Temperate. Intemperate. Total abstainer.

Occupation.

Primary Sore—hard, soft, doubtful.

Date of contraction.

Date of appearance. Duration.

Position.

Inguinal Glands—enlarged, indurated, suppurating.

**SECONDARY SYMPTOMS.**—Give dates of each.

Eruptions on Skin—character

Distribution

Affections of Mucous Membranes—patches or sores on mouth, tongue, tonsils, soft palate, pharynx, larynx, anus, vulva.

Eyes—iritis.

Other affections.

Ulcerative Lesions of Skin,

of palate,

of larynx,

of other parts,

of tongue,

of subcutaneous tissue,

Periostitis on tibia, clavicle, skull,  
other bones,  
Visceral Affections.

Affections of Nervous System.

**TERTIARY SYMPTOMS.**

Date of appearance after infection.

Nature, date, and duration of affection in

Skin.

Tongue.

Throat.

Eyes.

Nose.

Bones.

Testicle.

Viscera.

Nervous system.

Other parts.

*Treatment.*

Date of commencement.

Mercurial alone. Length of course.

Preparations employed.

If salivation. Date of first appearance.

Slight, moderate, severe.

Iodide of Potassium alone. Length of

course. Amount daily.

Mercury with Iodide of Potassium. Length

of course. Preparations and amounts

daily.

Simple non-specific treatment.

*If married.* Date of marriage.

Wife—healthy, infected. Date of infection.

Remarks regarding miscarriages or children

born within period of observation.

Remarks on any special feature of the case.

What is your opinion as to the prevalence of

Acquired Syphilis in your district?

## No. Va.

**INHERITED SYPHILIS:—**(1) *number of cases in infected families*, (2) *evidence of syphilis in the parents*, (3) *symptoms in children and result*, (4) *treatment*, (5) *prevalence*.

Observer's Name .....  
Address .....  
Date of first obs. ....  
Date of last obs. ....

(When in doubt about purport of any question refer to accompanying Memorandum for explanation.)

(Reply where possible by erasing words. This paper as soon as filled up to be returned to Secretary of Local Sub-Committee.)

Initials of child or case number.

Upper, middle, lower class. M. or F.

Born at full time or at what period.

**SYMPTOMS OF SYPHILIS.**

A. In Infant (before 2nd dentition). Age.

Date of first appearance of symptoms.

Nutrition—good, bad, earthy colour, snuffles, hoarseness.

*Eruptions:*

On face trunk scalp.

Nates palms soles.

Sores around mouth, anus, ears. Onychia.

Swellings of long bones near ends.

Enlargement of—liver, spleen.

Natiform skull.

Note character of vaccination scars.

Note any modification in the effects of vaccination attributable to syphilis observed in the child.

B. In Child (after 2nd dentition). Age.

Complexion earthy, natural.

Prominent forehead. Depressed bridge of nose.



Affections of cornea.  
 Scars around mouth, nostrils, anus.  
 Notched dwarfed upper median incisors.  
 Hearing impaired.  
 Periostitis, etc., of long bones.  
 Gummata—position of.  
 Ulcers—character and position of.  
 Stature weight.  
 History of syphilitic symptoms during infancy.

Remarks on any special feature of the case,  
 (e.g., rickets, etc.).  
 Further progress of case.  
 Plan of treatment.  
 How long has patient been under care of  
 observer?  
 Father—present evidences of syphilis.  
     past do.  
     Date of primary syphilis.  
     How acquired.  
 Mother—present evidences of syphilis.  
     past do.  
     Date of primary syphilis  
     How acquired.  
 Other confinements and children.

	State whether Abortion, Pre- mature Birth, or full-time child.	Date.	OFFSPRING.		Age of Child at Death.
			Infected.	Healthy.	
1st.					
2nd.					
3rd.					
4th.					
5th.					
6th.					

What is your opinion as to the prevalence of  
 cases of Inherited Syphilis in your district?

## THE FUNCTIONS OF THE LOCAL COMMITTEES.

THE work of organising the whole Association for the collective investigation of disease is being actively carried on. Several committees have now been appointed by various Branches of the Association, and others are being formed. Each Branch that has been applied to has most cordially responded to the call, and very able and active men have been enrolled to form local committees in the districts. It is evident that a large proportion of those most competent to aid in such a work as this will gladly welcome the opportunity of easily recording their cases of interest, and thus furthering the advance of our knowledge of medicine. The fact that the observations of so large a body of men are being simultaneously directed to the elucidation of certain obscure points, stimulates the desire of every man who takes an interest in his work to add his own experience and observations to the common fund. The committee supplies a necessity long felt; it gives voice to the great body of observers who are studying disease in all part of the country, and under varying conditions and surroundings; it will enable them easily and with little trouble to obtain answers to many questions which are constantly presenting themselves to the mind of every thoughtful practitioner.

At the present time, when these local committees are being asked for and appointed, some information concerning the work proposed for them may prove useful. To appreciate this, it is necessary to bear in mind the chief object for which the Collective Investigation Committee was appointed, namely, to advance our knowledge of medicine by means of the experience of disease gained outside the walls of the hospital.

The opportunities for studying medicine afforded by hospital practice are chiefly confined to observations on the later stages of organic disease, and to the study of acute diseases. Little or no opportunity occurs for the observation or recognition of the functional conditions which most probably precede organic disease. Some of the more important subjects concerning which it is hoped to gain information by the collective investigation of disease are included under the following heads:—

1. Facts are required upon various matters which can only be known to those who have opportunities of watching individuals, and sometimes families, for periods extending over many years, or throughout their lives. Of these, the following subjects are examples. *a.* The earliest symptoms and minor ailments which foreshadow the occurrence of grave constitutional conditions, such as rheumatism, gout, osteoarthritis, stone, goitre, etc.; or of organic diseases, such as cirrhosis of the liver, Bright's disease, phthisis, and many diseases of the cord and brain. *b.* The alliances of various diseases in families, and the sequence of disease in the individual: for example, the relation of syphilis to scrofula, the production of cancer by local irritation of a part or perverted function of an organ, the relation of gout to rheumatism and osteoarthritis, and of joint-affection to nerve-disease. *c.* The gravity of certain symptoms under varying circumstances, such as a mitral *bruit* in acute rheumatism, chorea, scarlatina, anæmia, pregnancy, or Bright's disease; of hæmoptysis in phthisis, gout, alcoholism, amenorrhœa, and other conditions; of albuminuria in Bright's disease, in scarlatina, in adolescence, in gout, etc.; of ankle-clonns and other reflex phenomena in functional and organic nerve-disease.

2. Information is required concerning the liabilities to disease in certain districts. *a.* The production, modification, or prevention of disease by variations in soil, elevation above sea-level, prevailing winds, amount of rainfall and sunshine, hygrometric condition of the atmosphere, and the qualities of drinking-water. *b.* The liability

ties to disease among the operatives in certain industries; this, though widely studied, is by no means worked out. c. The effects of syphilis on the population in districts where it is especially prevalent.

3. Many points of interest in the etiology of enteric fever, diphtheria, and others of the acute specific diseases, still await elucidation, and may well be undertaken by this committee.

4. The asserted communicability of phthisis is a subject which calls for immediate investigation.

5. The communicability of disease from animals to man is a question of increasing interest.

6. Last, but not least, carefully organised, extensive, and combined observations on the action of various drugs, may be expected to prove a fruitful subject for subsequent investigations.

These are some of the lines of work which the committee have in view; it remains to point out the manner in which the local committees may conduce to the work. The experience of past efforts of this kind, which have chiefly consisted in the circulation of schedules of questions, shows that they have failed on account of the complex and elaborate answers required, from the request being made for past and generally unrecorded experiences, from the want of a general interest in the subject, and especially from the want of an organisation by means of which personal application can be made to the members of the profession. It is hoped that, in the present movement, these causes of failure have been especially guarded against. The questions put to the observers are simple and definite; they can often be answered by a single stroke of a pen; they refer entirely to present or future observations, or to those of which a record has been previously kept; but the last and most important of these causes of failure will be met, it is thought, by the local committees. These will have essentially a stimulating function. Each member will be expected to interest his friends and neighbours in the work, and, by personal application to them, it is believed that he will obtain their assistance. The primary function of all local committees should be personal application to the members in their district, and this should be carried out in a carefully organised and systematic manner; each member of the local committee undertaking to make personal application to certain selected persons, for whom he should be made responsible.

The best method of organising and working each Branch will vary greatly; and this, again, can only be determined by the local committee.

In addition to propagating the work in their respective Branches, the local committees will be asked to consider the question of the prevalence of particular diseases in their districts; indeed, all the subjects of inquiry in the second group will come under their notice. Not only should the influence of climatic conditions in the production and prevention of disease be studied by them, but also the diseases produced by special trades or manufactures, and the local outbreaks of specific fevers. It is probable that, in many instances, special investigations, of local interest, will be suggested and carried out by local committees. Whenever a local investigation is proposed, it is hoped that a scheme will be drawn up and the necessary questions framed, by the local committee; and that it will then be submitted to the general committee for criticism and suggestions. In drawing up the cards about to be issued, it has been found that those who frame a scheme of this sort, and propound the questions in the first instance, are never able to effectively criticise their own work. This function of criticism is exceedingly well filled by the central committee, upon which all shades of thought and opinion, as well as much special knowledge, are represented.

The general committee will always be glad to lend assistance that may be necessary in carrying out any local investigation, either in organising, printing, or secretarial work.

Finally, each local committee is invited to send to the general committee a representative who will not only represent the Branch, but will keep the local committee in communication with the central body. Though it may be impossible in many cases for these representatives to attend the meetings in London, nevertheless the subjects to be brought forward at these meetings will always be submitted to them for remarks and criticisms, which can be made by letter, and laid before the general committee.



The three cards which have been prepared, after many revisions and alterations, are now being stereotyped. Specimen slips showing how they should be filled up will be sent with each. The process is necessarily a long one, and some unavoidable delays have occurred, but they will be ready for distribution by the second week in May : together with reprints of the memoranda on the subjects of investigation, which will be issued to those who may wish to possess them. The cards will be issued by the local committees, or by the secretaries appointed for the various districts, to whom application should be made by those willing to take part in the work.

THE  
COLLECTIVE INVESTIGATION  
RECORD.

EDITED

*BY THE COLLECTIVE INVESTIGATION COMMITTEE  
OF THE BRITISH MEDICAL ASSOCIATION.*

PROFESSOR HUMPHRY, M.D., F.R.S.,  
CHAIRMAN OF THE COMMITTEE.

VOL. II.

---

**JULY, 1884.**

---

PUBLISHED BY THE  
BRITISH MEDICAL ASSOCIATION, 161A, STRAND.

# ALLEN & HANBURY'S

## "STANDARD" HYPODERMIC SOLUTIONS

### UNIFORM. ACTIVE. PERMANENT.

These Solutions are made to a Standard Dose of 5 minims, and comprise:—

Acetic Acid, Aconitine, Ammonia, Apomorphine, Arsenic, Atropine Salicylate, Atropine Sulphate, Atropine and Morphine, Caffeine, Chloral, Conine, Curare, Daturine, Digitaline, Digitaline and Atropine, Digitaline and Morphine, Ergot (purified), Eserine Salicylate, Gelsemine, Homatropine, Hyoscyamine, Mercury Bichloride, Morphine Acetate, Morphine Sulphate, Picrotoxine, Pilocarpine, Quinine, Strychnine, Veratrine.

**CHRISMA.**—A pure and neutral solid hydro-carbon. Forms a perfect ointment base, and simple dressing.

**CHRISMA ALBUM.**—Bright and colourless, and entirely free from Smell and Taste.

**CHRISMA SULPHURIS.**—Superior to Ung. Sulphuris, being an actual *Solution* of Sulphur in Chrisma. By this the application of Sulphur to the skin is more effective, and it is neither irritating, offensive or unsightly. An effective Parasiticide.

**TRUE (PRECIPITATED) OLEATES OF THE METALS.**—In three forms: Pure Oleate, Dusting Powder (10 and 20 per cent.), and Ointment (20 per cent.).

These Oleates furnish the most efficacious medication for Skin Diseases.

Oleates of Aluminium, Arsenic, Bismuth, Copper, Iron, Lead, Mercury, Nickel, Silver, Tin, Zinc, &c.

*Price List of ALLEN & HANBURY'S Pharmaceutical Preparations, Specialities, and General Drugs and Chemicals sent free on Application.*

ALLEN & HANBURY'S, Plough Court, Lombard Street, London, E.C.

## SCHIEFFELIN'S

Are made of the best materials; perfectly protected; entirely and quickly soluble; coated while soft; not subjected to the injurious effects of heat in the process of manufacture. The mass is worked to the highest possible degree of uniform intermixture, and subdivided with scrupulous exactness. The coating is an inert, tasteless, and odourless compound, which is so transparent, colourless, and thin, that the distinctive colour of each pill is revealed through the coat. No sub-coating is used. The solubility of the coating is not impaired by age.

## SOLUBLE

Our list comprises about 400 varieties of Formulæ, which, with prices and samples, may be had on application to

## PILLS.

ALLEN & HANBURY'S, Sole Agents for W. H. SCHIEFFELIN & Co., New York.



# CONTENTS.

---

	PAGE
REPORT OF THE COMMITTEE . . . . .	1
REPORT ON ACUTE PNEUMONIA:—	
I. ON EPIDEMICS OF PNEUMONIA . . . . .	5
1. British . . . . .	5
2. Foreign . . . . .	10
II. REPORT ON THE RETURNS . . . . .	27
NOTE ON THE ETIOLOGY OF PNEUMONIA . . . . .	60
GENERAL CONCLUSIONS . . . . .	64
NAMES OF REPORTERS AND NUMBER OF CARDS RETURNED	
BY EACH . . . . .	72
INDEX . . . . .	75
ORIGINAL COMMUNICATIONS:—	
[ <i>The Committee is not responsible for the statements or opinions contained in the papers under this heading.</i> ]	
ON AN EPIDEMIC OF PNEUMONIA IN THE PUNJAB. BY SURGEON-MAJOR S. E. MAUNSELL . . . . .	77
ON A SECOND EPIDEMIC OF PNEUMONIA IN THE PUNJAB. BY SURGEON- MAJOR S. E. MAUNSELL . . . . .	93
ON AN EPIDEMIC OF PNEUMONIA IN GLASGOW. BY JAMES FINLAYSON, M.D., PHYSICIAN AND LECTURER ON CLINICAL MEDICINE TO THE WESTERN INFIRMARY, GLASGOW . . . . .	100
MEMORANDUM ON THE INCIDENCE OF FATAL PNEUMONIA. BY G. B. LONGSTAFF, M.A., M.B. OXON., M.R.C.P. . . . .	102
ON THE MICRO-ORGANISMS OF PNEUMONIA. BY GEORGE M. GILES, M.B., F.R.C.S., SAN. CERT., LOND. . . . .	106

---

	PAGE
FIRST REPORT ON PUERPERAL PYREXIA . . . . .	118
APPENDIX OF RETURNS ON FEVER IN THE PUERPERAL STATE . . . . .	136
ORGANIZATION FOR THE COLLECTIVE INVESTIGATION OF DISEASE . . . . .	193
MEMORANDA AND CARDS ALREADY ISSUED . . . . .	199

THE

# COLLECTIVE INVESTIGATION RECORD.

---

## REPORT OF THE COLLECTIVE INVESTIGATION COMMITTEE.

THE following is the Report of the Collective Investigation Committee, which was presented to the Council of the Association, to be laid before the Annual Meeting of the Association at Belfast.

The work of the Committee has been carried on during the past year with much activity, and with an encouraging amount of success.

In October last, Dr. Mahomed resigned the office of Secretary, but continues to give his assistance as Honorary Secretary; and on the nomination of the Committee, Dr. Herringham has been appointed Secretary by the Committee of Council, and has continued the work with energy and success. In consequence of the absence of Dr. Herringham, with the sanction of the Committee, during the last three months, the secretarial work, which has been very heavy, has devolved upon Mr. Oswald A. Browne; the Committee have to express their obligation to him for the careful and unremitting attention which he has given to it.

As the expenses of carrying on the work appeared to be increasing and uncertain, the Finance Committee thought the interests of the Association and provision for the work would be best met by allowing a grant for the year to the Committee of £600; this to include the salary of £200 to the Secretary, as



well as the £100 for secretarial and travelling expenses, already granted by the Association. The remaining £300 is to meet the expenses of printing cards and memoranda, the payment for literary work done, the printing and publication of the RECORD, &c.

The sum may seem large, but the work done for it is fully proportionate; and this bears little proportion to the great amount of pecuniarily unrequited work which is done by the many members of the profession who are endeavouring, through this Committee, to promote the science and practice of medicine and to add to the reputation and good influence of the Association.

The first number of the COLLECTIVE INVESTIGATION RECORD, referred to last year, was issued in July. It contained a complete Report, prepared on behalf of the Committee by Dr. Burney Yeo, on the replies that had been received in answer to the inquiry on the Communicability of Phthisis; the replies themselves being carefully edited, classified, and published for reference. The Committee may refer with satisfaction to the numerous references to this report which have been made in the current medical literature, and especially by Dr. Andrew, in his Lumleian Lectures at the College of Physicians, where they are described as—

“The largest collection of observations upon the communicability of phthisis among men, and by far the most important, inasmuch as it includes communications from men of large experience and of every shade of opinion, is that for which we are indebted to the Collective Investigation Committee of the British Medical Association. The report, based by the Committee, upon the facts submitted to them, evidently leans to the contagionist side; but the premises are so faithfully given, that it is easy for anyone to judge for himself how far the conclusion drawn from them is correct.”

This number of the RECORD further contains preliminary Reports on Pneumonia, Chorea, Acute Rheumatism and Diphtheria; each of which has distinctly advanced our knowledge of the subject under inquiry.

The favourable manner in which this first number of the RECORD has been received, and the recognition of the intrinsic value of its contents, encourages the Committee to issue in July, of this year (1884), a second number containing a Report, prepared on behalf of the Committee by Drs. Sturges and Coup-

land, on Pneumonia, based on the records of more than a thousand cases which have been received by the Committee. This Report, on probably the largest number of cases ever submitted to analysis, cannot fail to interest the profession and throw light on the clinical history of the malady.

The number contains also a preliminary Report on nearly four hundred cases of Puerperal Pyrexia, each of which will appear in abstract. The Report, in addition to the information given, furnishes valuable indications for further inquiries. These Reports, together with some original articles of much interest on Pneumonia, and the cards and memoranda issued during the year, form a volume of more than three hundred pages, which is issued to the Members of the Association at the price of 1s. 6d.

The subjects at present under investigation, in addition to Pneumonia, Chorea, Rheumatism, Diphtheria, Syphilis, Gout, and Puerperal Pyrexia, are :—

1.—The habits, maladies past and present, and family history, of PERSONS WHO HAVE ATTAINED GREAT AGE.

2.—The Clinical Features and Causation of PAROXYSMAL HÆMAGLOBINURIA.

3.—The significance of ALBUMINURIA in apparently healthy persons.

4.—SLEEP-WALKING, the subjects in whom it occurs, their liabilities to nervous disease, and their responsibility for acts committed during somnambulism.

In proposing these four subjects, the Committee have been influenced by the desire to ascertain particulars and preserve records of maladies concerning which little is known, and which are so rare that the experiences of any individual goes for little, and which, therefore, especially demand collective inquiry. At the same time the efforts of the Committee are mainly directed to the furtherance of our knowledge of diseases which are more frequent and therefore more important.

The Committee feel that the thanks of the Association are due to the many gentlemen who have, by their returns and in other ways, contributed to forward this important work during the past year. They have to regret the loss by death of the following gentlemen, who have rendered much valuable assistance as local Secretaries, viz.: Dr. Hudson, of Redruth; Dr. Burt, of

4 *REPORT—COLLECTIVE INVESTIGATION COMMITTEE.*

Kendal; Dr. E. S. Scott, of Shrewsbury; and Mr. Kirby Smith, of Northampton.

The Committee are glad to be able to add that Sir William Gull has acceded to their request that he should bring the subject of International Collective Investigation of Disease before the forthcoming Congress at Copenhagen. A general meeting of the Congress has been devoted to this purpose, and it is hoped that a wide-spreading international organisation may be established.

(Signed) G. M. HUMPHRY,  
Chairman of the Committee.

July 2nd, 1884.



## REPORT ON ACUTE PNEUMONIA.

### PART I.

#### ON EPIDEMICS OF PNEUMONIA.

---

THE following short notice relative to the several outbreaks of pneumonia in this country and abroad, which have given support to the belief that this affection may occur, not only as an epidemic but sometimes as an infectious disorder, will form, it is thought, a suitable prelude to this Report.

#### I. BRITISH.

As regards Great Britain, only a brief summary need be given; all that is of importance relative to epidemic and contagious pneumonia has been recorded within the last ten years, a period which may be taken to represent the time that such forms of the disease have attracted special notice in this country. We may refer to them under the following headings:—

1. *Epidemic pneumonia*, occurring in districts or houses after the manner of epidemic catarrh.

2. *Pythogenic pneumonia*, occasioned by sewer gas, fœcal accumulation, over-crowding, etc., and apt to affect several individuals so exposed.

3. *Infectious pneumonia*, imported into a house or district and spreading from person to person.

#### 1. *Epidemic Pneumonia.*

The prevalence of pneumonia at certain seasons, whether alone or in conjunction with epidemic catarrh, or herpetic fever, only needs mention in this place in order that it may be defined and separated from the two forms of the disease just mentioned, which, although of different origin, are also apt to be epidemic.

Dr. Couldrey, of Scunthorpe, has reported several such epidemics (*Lancet*, Nov. 16, 1878), and (inasmuch as he has been good enough to send us details of two of them) these may be quoted as examples. Scunthorpe is a new district, having a soil of sand and slag. It contains between 2,000 and 3,000 inhabitants, nearly all engaged in the iron industry, crowded together in ill-built houses. The village is subject to epidemics of measles and scarlatina, but enteric fever is extremely rare.

"It is the habit of pneumonia," says Dr. Couldrey, "to occur in this district in spells, with long intervals of rest, when there is hardly a single example of it." He gives the particulars of two such visitations. With the first, no other epidemic prevailed; with the second—there being an interval of two months between—erysipelas was prevalent, and later on scarlatina. The first series extended from the middle of October to the middle of January, 1881–82, during which time 18 cases of pneumonia occurred, six of them being children—two died. The second series began in the following March and lasted two months. Seven cases were attended by Dr. Couldrey, of whom two died.

We have here but one illustration amongst many of pneumonia occurring after the manner of the more familiar epidemic catarrh, but not in conjunction with it, nor yet associated with any illness of pythogenic origin. The occurrence of pneumonia after this manner stands apart from other forms of so-called epidemic pneumonia, and this particular illustration has been selected owing to the extreme rarity of enteric fever in the same locality.

## 2. *Pythogenic Pneumonia.*

In the spring of 1874, pneumonia broke out in a boys' school at East Sheen, five persons being attacked, of whom one died.

The circumstances are reported as follows in the *Medical Times and Gazette*. On the 14th of March, in spite of urgent protest on the part of the school authorities, the parish sewer opposite the house was opened for the purpose of ventilation. The ventilator was placed in the high road and provided with the supposed protection of a charcoal filter. On the

20th of March, the occurrence of an unusually high tide placed the mouth of the sewer for a time under water. As a consequence the sewer gases forced their way through the gravel covering of the ventilator and it was necessary to close all the windows of the upper rooms which overlooked it.

The day following the gas escape, a boy occupying one of these rooms was taken ill of pneumonia, and the same evening two other boys. Two servants of the house were likewise affected, one of whom died. It should be added that for 15 years this house had been remarkably healthy, that its drainage was perfect, and that on the closing of the sewer no further case of illness occurred.

Not long after the publication of these particulars, Drs. Grimshaw and Moore drew attention to the prevalence of pneumonia of supposed pythogenic origin in Dublin, during the spring and summer of this same year, 1874. The authors sought to distinguish their cases from legitimate pneumonia, pointing out that in some instances it was "rather congestion than inflammation of the lung, having a close relationship to enteric fever." "The severer forms, however," it is added, "followed nearly the course of true pneumonia." Owing to the difficulty of separating their cases from enteric fever on the one hand, and true pneumonia on the other, the writers were unable to fix precisely the mortality of pythogenic pneumonia, but it did not appear to be usually fatal. It is important to observe that in the one instance, where post mortem examination was made, the lung presented the same appearances as in ordinary pneumonia. Clinically, however, the authors were disposed to distinguish it therefrom, owing to its extremely sudden invasion, the frequency with which the disease was arrested at an early stage, and its being less liable to attack the lower lobe of the right lung.

In an appendix to their paper, Drs. Grimshaw and Moore record eight examples of houses exhibiting excessive neglect in respect of drainage and accumulation of ordure, in four of which cases of pneumonia had occurred.\*

\* Reference may here be made to Dr. Gooch's account of an outbreak of pneumonia at Eton, due apparently to drain defect, reported in our Preliminary Report p. 106.



### 3. *Infectious Pneumonia.*

Of the evidence afforded by our literature in support of the existence of an infectious form of pneumonia the following illustrations may be quoted, both recent and both fully and ably reported. We can here give but short extracts, referring the reader to the original papers. The first is by Dr. Patchett. A family of five elderly people, four brothers and a sister, lived together on a steep hillside, with good sanitary surroundings and apart from any known epidemic or septic influence, the men well-to-do farmers. On the 13th of January, 1876, James, the eldest, was taken ill and died in six days with typical pneumonia. The day before his death, John, the next in age, had a rigor, and three days after his brother he also died of pneumonia. The day after John's death the two remaining brothers both suddenly got pneumonia and both speedily died of it. There was now but the sister left. She had nursed her brothers throughout "and kept her health remarkably well." But the day after the death of the two last, she also was seized with pneumonia of the right lung and died in three days. Thus, in less than a fortnight, the entire family were swept away by a disease which the narrator describes as "typical pneumonia."

The second illustration is by Dr. Daly, and is hardly less striking. A child of the family concerned was ill and feverish, but no very definite symptoms were noted, and he soon recovered. Nine days later, however, two other children were seized with acute pneumonia. On the fourth day of their illness the mother, a lady, who was constantly in the sick room, took the same disease, as well as the youngest boy. With four undoubted cases of pneumonia in the same house within a few days, it occurred to Dr. Daly to examine the child who first of all had showed signs of illness. He satisfied himself that this child too had suffered from pneumonia. In the later part of her illness, the mother of the lady came to nurse her daughter. The latter died, and six days later the mother also died, and of her daughter's disease.\* The sanitary condition of the house was

\* Reference may here be made to Dr. Gilbert Child's report of an outbreak of pneumonia, presumably infectious, in our Preliminary Report, p. 105.

most carefully investigated, and no fault could be discovered; there was no epidemic prevalent, and none of the patients had been out or otherwise exposed. Further, it was noticeable that those members of the family who were habitually together were all attacked; the rest escaped.

In connection with such narratives it is pertinent to refer to some experiences of Dr. C. Budd, quoted in a paper on "Infectious Forms of Pneumonia," by Dr. Wynter Blyth.

A farmer, with pneumonia, was nursed by his niece; she became affected with the same disease and carried it to her husband. Again, an old man, with pneumonia, reposed on the breast of a relative for the greater part of his illness. The relative was afterwards affected in the same way.

Further, and upon the authority of a practitioner whose name is not given, Dr. Blyth relates the following. "A farmer took pneumonia and died. A week later his servant took it and went home, where she communicated the same disease to a married sister."

Examples of a like description will be found scattered through this Report; they are summarised in Abstract XIV., and an analysis of such cases is given on page 58.

OCTAVIUS STURGES.

#### BIBLIOGRAPHY.

---

1. COULDREY.—'Lancet,' November 16, 1878, and personal communication.
  2. 'Medical Times and Gazette,' 1874, vol. i., May and June.
  3. GRIMSHAW and MOORE.—On Pythogenic Pneumonia. 'Dublin Medical Journal,' May, 1875.
  4. PATCHETT.—'Lancet,' 1882, vol. i., p. 305.
  5. DALY.—'Lancet,' 1881, vol. ii., p. 824.
  6. WYNTER-BLYTH.—'Lancet,' Sept. 18, 1875, vol. ii.
-

## II.—FOREIGN.

RECORDS of epidemics of genuine pneumonia are not very numerous in literature, and it has been already stated that they fall mainly within the last decade. But for three hundred years (Hirsch, Haeser) epidemics have occurred, and been recorded, in which pulmonary symptoms have prevailed, although it is impossible to say whether they were what would now be called cases of uncomplicated pneumonia or not. Indeed, it is highly probable that many of these epidemics were part of other diseases, as typhoid fever and influenza, and therefore do not strictly fall within the scope of the present review.

Thus *Hirsch*\* (1), in speaking of pneumonia, after a lengthy discussion upon the meteorological and climatic conditions that influence its prevalence, states that a large number of cases of primary pulmonary or thoracic inflammation are recorded, which can be explained by no known etiological factors; and for the occurrence of which some unknown influence or specific cause, miasmatic or other, has to be invoked. Such cases are wont to occur in epidemic form, and have been variously described as 'bilious' or 'putrid pleuropneumonia' or 'typhoid pleuropneumonia.' Hirsch appends a chronological table, which includes references to records of no fewer than 187 such epidemics, from the 16th century to the middle of the 19th. It is noteworthy that Italy furnished the larger proportion in the 16th and 17th, France in the 18th, and N. America in the 19th centuries. Only six of the epidemics are assigned to the British Isles, viz., one in February, 1736, in Fife (*Edinb. Med. Observation*, v., 35); two in Plymouth, spring, 1740, and winter and spring, 1745-6 (recorded by Huxham†); one in London, in January, February, 1805 (Bateman, *Report on Diseases of London*, 1819, p. 37); one in Dublin in 1832-3 (Hudson, *Dublin Journal of Med. Sci.*, vii., 372, in an article entitled

\* The second edition of this remarkable work has not yet been completed, so that we are unable to give Professor Hirsch's matured opinion upon the subject of pneumonia. It is matter for congratulation that the work will now be available for English readers in the translation by Dr. Creighton (New Syd. Soc.).

† Huxham was the first to employ the term "pneumonic fever" to this type of the disease.



"Observations on Typhoid Pneumonia"), and one in 1856 in Leicestershire (by Barclay, *Association Medical Journal*, 1856, June). Hirsch points out that these numerous observations, gathered from all quarters of the globe, include many varieties of the disease; and he is struck by the fact that so large a proportion, especially those of later date recorded in America and Switzerland, coincided or followed upon the prevalence of typhoid fever. This remark justifies the caution expressed above, as to admitting into the records of simple epidemic pneumonia instances of what were possibly typhoid fever with pneumonic complication. He quotes from Low (*American Med. and Philosoph. Register*, 1813, iv., 31) the following passage:—"The causes of this disease (epidemic pneumonia) are probably the same as typhus gravior and pneumonia, along with a peculiar constitution of the atmosphere, apparently beyond the reach of our investigation. As far as I have observed, no class of persons are exempt from it; it attacks equally the opulent and the indigent, the temperate and the intemperate, but proves much more fatal to the poor and intemperate, frequently from the want of necessaries to the one, and the exhausted constitution of the other." It should be added that Hirsch discriminates between those cases of typhoid pleuropneumonia and the malarial form, which is recorded mainly by American authors, and which refers to epidemics of pneumonia in regions where intermittent fever prevails.

*Haeser* (2), who notes that "pneumonia maligna," or "typhous pneumonia," was differentiated as distinct from other forms of pestilence as early as the 16th century, also points out the intimate connection of many epidemics of this disease with those of typhoid fever, and considers that the testimony is very strong in regarding "typhous pneumonia" as a special form of typhoid fever. He also alludes to its relations with exanthematic typhus, and the frequency with which typhous pneumonia occurs in mountainous regions. He quotes Guggenbuhl, who states that the "Alpenstich," which occurs in the Swiss mountains in spring, and which has occasionally spread widely, has been known for an inconceivable time, and is attributed to the Föhn, the "Sirocco of Switzerland," whereby the snow and ice masses of the Alps are converted into moun-

tain torrents. Similar conditions have, he says, been observed in connection with the frequent spread of typhous pneumonia in upper Italy and perhaps also in Peru. Some epidemics of this kind were observed as early as the 13th and 15th centuries, and Colle regarded the "black death" as a malignant and contagious pleuropneumonia.

Leaving aside these historical researches and coming to the modern records of epidemics of pneumonia, we find that they are very varied in extent and character. The whole subject has been lately reviewed by *Mendelsohn* (3), to whose essay we may here express our indebtedness for many of the facts that follow. In the majority of instances we have referred to the original writings, but in others we are obliged to avail ourselves of this admirable summary. *Mendelsohn* divides the records into three groups, viz., those which deal with more or less widespread epidemics, those limited to large buildings (as prisons and barracks), and those confined to houses. Instances are not few of each of these, and we may profitably adopt a similar arrangement here. Another writer, *Sanders* (4), also reviews the subject with considerable fulness; and indeed there is no lack of general literature which mostly goes over the same ground, in reproducing the same instances.

An epidemic of great severity occurred in Iceland in the year 1863, and is recorded by *Dr. J. Hjaltelin* (5), who, although he had practised in the country for ten years had not hitherto met with pneumonia except in the sporadic form. The epidemic began in the winter at the northern part of the island, and it is noticeable that the occurrence of pneumonia was preceded by an epidemic of influenza. The writer does not give details as to the precise extent of the epidemic, and it is probable that owing to the small number of medical men in Iceland, such details could not be supplied. He states, however, that in January and February, 1863, cases of genuine pneumonia became very frequent, and 80 of these came under his personal observation, nine proving fatal. The period was one of severe storms, and a dry and cold air impregnated with ozone, to the irritant effects of which he attributes the great mortality from respiratory disease in the northern latitudes, and the frequency of pneumonia amongst the Eskimos. The paper is, however,

mainly devoted to a discussion of treatment by calomel, and by blood-letting, against which latter practice Hughes Bennett was then turning the current of opinion.

An account of an interesting epidemic of typhoid (or "pythogenic," as it might more strictly be called,) pneumonia, which occurred in Florence and other parts of Tuscany in the winter and spring of 1877-78, is given by *Banti* (6). The disease began towards the end of the autumn, and gradually assumed epidemic proportions. Typhoid fever was prevailing at the time, and this disease increased in the following summer, when the pneumonia epidemic had subsided. All classes of inhabitants were attacked, young and old, weak and strong, rich and poor, and owing to the peculiar type of the disease, diagnosis was often very obscure, sometimes not correctly made until post-mortem examination revealed the true nature of the case. Some idea of the extent of its ravages may be gleaned from Banti's statement that on one day out of ten cadavers he examined, nine were cases of pneumonia. In some cases the onset was preceded by bronchial catarrh; in others it was abrupt; but in most there was a prodromal period of malaise, headache and prostration; whilst during the course of the fever the signs of pulmonary inflammation were often entirely absent. In some cases pneumonic signs appeared as late as the seventh or eighth day, and in others the inflammation spread from one part of the lung to another. It was of the asthenic typhoid type with high fever, and in the cases of recovery this took place by *lysis*. But the ordinary termination was in death, mainly from cardiac paralysis, and Banti made a large number of the post-mortem examinations. In addition to pneumonic hepatization and hæmorrhagic congestion, the main features of the morbid anatomy of these cases were a dark colour and fluidity of blood, fatty degeneration of the cardiac muscle, swelling and softening of the spleen. But in no case were any of the intestinal lesions characteristic of typhoid fever met with. The record is particularly valuable in that it indicates anatomically a specially severe and malignant type of pneumonia. Etiologically, the disease could not be attributed, he says, to atmospheric influences, nor to insanitary conditions alone, on account of the wide area over which the disease



spread, and the varied social status of the sufferers. At the same time the coincidence of typhoid fever cannot be entirely overlooked, and although Banti believes that this epidemic was a peculiarly infectious form of pneumonia, he hesitates to deny any connection between it and typhoid fever.

In a paper dealing with the etiology of pneumonia *Moellmann* (7), of Simmern, notices a few small "epidemics" in a region where the disease seems to have been generally very prevalent. In an experience of about 12 years (1868-79) he had met with 220 cases in Simmern and its vicinity, a somewhat elevated region between the Moselle and Rhine, and subject to rather severe climatic changes. He details the monthly prevalence, which shows that most cases fall in February, and that the three winter months (December, January and February) had nearly twice as many as the spring months, three times as many as the summer, and four times as many as the autumn. The disease often broke out in a village in small groups of cases, and he records three such "epidemics," viz.: 1. In R——, 300 inhabitants, five attacked in 18 days (February, 1873), all recovered. 2. In M——, 500 inhabitants, seven cases within 17 days (May, 1873), all recovered. 3. In K——, 500 inhabitants, eight cases in December, 1874, three deaths. These epidemic cases were marked by considerable extent of the local inflammation, high fever, and a tendency to early cardiac failure.

Mention may here be made, although full details are wanting, of an observation by *Feldhausen* (8), in Ebstein's clinic at Göttingen. Twenty-five cases of pneumonia occurred in one district, five coming from one street, of whom two were doubtless in the same house, the second patient falling ill three days after the first. In another street there were four cases in two houses, and cases continued to occur at intervals. In three other instances, three patients, and in two others, two patients came from the same house.

A very complete account of one of these village epidemics, which form the main part of the literature of the subject, is given by *Butry* (9). It occurred in 1881 in Becherbach (Meisenheim). The winter had been severe, with N.E. winds, but there had only been a few cases of pneumonia, and those of a mild type;

but in the spring a malignant epidemic broke out. Out of a population of 460, 20 persons, *i.e.* 4 per cent., were attacked within a few weeks. The first fell ill on March 26th, the next in the second week of April, two in the third, and ten in the fourth week of April, one in the second week of May, two in the third, and two in the fourth week, the last case occurring on June 17th. Ten cases, *i.e.* 50 per cent. of those attacked, died!; eight of the fatal cases being amongst the 12 individuals over 15 years of age. The disease spread, mainly amongst relatives and neighbours, and those who visited the sick at their homes. Thus No. 3 was the cousin of No. 2; No. 4 the brother of No. 3; No. 5 brother and neighbour of No. 12; No. 9 sister of No. 10; No. 14 was a relative of No. 12, and sister to No. 15, neighbour to No. 19, and also related to No. 12; No. 16, mother-in-law of No. 13, occupying the same house; No. 20 lived in a house opposite to Nos. 5 and 12; and Nos. 6, 7, 8 and 14 were all neighbours. The disease was of the asthenic type. In seven cases the upper lobe was involved, and in five the pneumonia was bilateral. There was no splenic swelling. Convalescence was protracted. Jaundice occurred in many of the fatal cases.

We have only met with a brief notice of the following observations made in 1881 by *v. Holwede*, of Brunswick, and *Mün-nich*, of Ober-Sikt (10), which were made as a contribution to the doctrine that croupous pneumonia is an infectious disease. In the small village of Ober-Sikt of 400 inhabitants, 15 children between the ages of one and five years (or 30 per cent. of whole number) were attacked with pneumonia within the space of thirteen days. In one house there were three cases; in another two. Cold could not be invoked as the cause of the outbreak for most of the children had been confined within doors for some weeks during the cold winter.

The epidemic which is recorded by *Penkert* (11) is one that is very well known, for it has been quoted very largely. It took place from March 28th to May 28th 1881, in the village of Rietnordhausen, seven kilometres from Artern. Out of 700 inhabitants, 42 were attacked with the disease, and all but three of these were children below 12 years of age. The three adults apparently contracted the disease from the children. Fifteen were males and 27 females. There were four deaths. The cases

were mostly typical, some abortive; and there is nothing in the symptoms requiring special mention. Penkert gives brief details of each case. The epidemic began amongst the children attending the school, and 12 of them fell ill before the Easter vacation. No case occurred during the holidays from April 13th to 28th, when again another of the school children was attacked. After that the epidemic spread more widely, and many instances of apparent contagion were observed with an incubation period of from five to eight days. It appears that the village is on the side of a hill, and the school buildings at the lowermost part, which is generally flooded in the spring time. The buildings had been erected four years before, and lay to the east of a graveyard, the village street intervening; at the north end of the graveyard was the church, whilst cattle sheds and stables partly surrounded the yard on the west-side; a stagnant pond intervening. The explanation of the outbreak, under these conditions, given by Penkert is highly probable. The churchyard, pond and stables, and a season when the level of subsoil water was unusually high, were elements well fitted to generate a specific poison. At any rate the N.E. wind, which set in a few days before the first child sickened, must have conveyed such emanations as were present directly across the graveyard to the school buildings.

In a volume issued last year under the editorship of Professor Juergensen, the foremost advocate of the constitutional nature of acute pneumonia, there is an admirable and exhaustive account of an epidemic which occurred at Lustnau, in 1881, by *Scheef* (12). Lustnau is a village of 1633 inhabitants, and 233 houses; and has been medically under the charge of the Tübingen Policlinic since October, 1873. Owing to this a comparison was readily made between previous years and 1881, as to the prevalence of pneumonia in the place. From October, 1873, to the end of 1880, there had been 143 cases of the disease, the largest number in any year being in 1878, when there were 29 cases. From October to December, 1880, only four cases occurred; but at the beginning of 1881 an epidemic broke out, so that during January there were 19 cases, a number equalling the total amount occurring in 1880. In February, 10 cases occurred, and in March, April and May, 5 each month, *i.e.*, 2·7 per cent. of the



population, or 17·5 per cent. of the total number of cases of disease in the same period. The first 29 cases occurred within 56 days, after which there was an interval of 14 days till March 12th, when a recurrence of the epidemic broke out, but less severely. Between May 23rd to the following November no case of pneumonia occurred in Lustnau. The majority of those attacked were adults, and 60 per cent. were males. The disease broke out simultaneously in many houses, and spread over the whole village, prevailing more in some parts than in others; and in some houses more than one case occurred. Thirty cases occurred in houses which had in previous years contained cases of pneumonia. The rainfall during the period over which the epidemic lasted was comparatively small; and the winter had been the mildest since 1874. The mortality rate was high, 10 deaths, or 23 per cent. In all the fatal cases (full details of which are given) there was pleuritis,—in some purulent; in many pericarditis; and in many subserous and submucous hæmorrhages; also parenchymatous degeneration of the liver, spleen and kidneys. In 26 of the cases one lobe of the lung was involved; in 11 two lobes; in three both lungs, one fatal case presenting grey hepatisation of the whole of both lungs. In no fewer than 23 cases was the upper lobe attacked, and 15 of the cases were of the spreading or “migrant” form. In no single instance could exposure to cold be adduced as the cause of the illness; and in many cases the attack was preceded by prodromal symptoms. The onset was marked by definite signs in 17 cases, by chilliness, &c., in 13. Ten of the cases terminated by *lysis*.

Zang, a village of 549 inhabitants, seated on the Aalbuch, and surrounded by woods, was the seat of an epidemic of pneumonia in 1882, which is recorded by *Schmid* (13), of Königsbrunn (Wurtemberg). Pneumonia was rather a common disease in the place, which is mainly inhabited by the labouring class, accustomed to rude fare. The dwellings are detached, and although the water-supply is bad yet typhoid fever is rare. Nor did relapsing fever, which, as in the case of typhoid, has been considered by some to occur when pneumonia prevails, ever occur. The first to fall ill was a youth eleven years of age on April 5th; the next case, a girl of sixteen, was attacked on May 11th, and

from that date to June 19th 17 other cases occurred, mostly in houses near to one another. In one instance six members of a family of eight fell ill in succession, including an infant who died, four children from three to eleven years of age, and the father. In another house a woman 68 years of age was taken ill on May 24th. She died, and her two sons, aged 42 and 44 years, living near her, also fell ill on May 29th and June 1st respectively, and both suffered severely. Two out of the 19 cases were fatal, and in four the signs were abortive. The early part of May was warm, and then cold weather set in till the 21st, when it became warm again. These changes of temperature may have increased the tendency to the disease.

The last of these village epidemics we have to record is one reported by *Senfft* (14), of Bierstadt (Wiesbaden). It occurred at Erbenheim, a well-built place, situated at a moderately high elevation, and containing 1500 inhabitants. For more than five years the village had been free from typhoid; and the cases of pneumonia amounted to only 4 per cent. of all diseases. Within the space of three weeks, from November 2nd to November 24th, 1882, 59 cases of pneumonia occurred, the daily number rising to seven on the 11th, falling to two and three on the next three days, and rising again to six on the 14th, after which it more or less steadily declined. On comparison with meteorological conditions *Senfft* found that the course of the epidemic was precisely opposed to the general experience that pneumonia is favoured by northerly and easterly winds and dry weather. For the numbers attacked on each day varied conversely with the rise and fall of the barometer; and more cases occurred with the S.W. wind than with N. and E. Too much stress should not, however, be laid on this, since the period over which the epidemic lasted was so brief. Of the 59 cases (24 males) only 13 were over 10 years of age, and but five died, *i.e.*, 2·95 per cent. Thirteen cases were left-sided; 20 right-sided; and 20 bilateral; in many the inflammation began at the apex. Pleurisy occurred in most of the cases; pericarditis in none. Diarrhœa was present in one-fourth of the whole number; but in no case was any swelling of the spleen made out. The average duration was 11·7 days; crisis occurring in most from the third to the eighth day. *Senfft* thinks the contagious nature of the

affection can hardly be doubted, for particular streets were selected, and especially neighbouring houses. Again, two cases occurred in 10 families, three in six families, and one in 21 families. Another interesting fact is that after this epidemic many cases of influenza appeared in the district, and especially at Erbenheim.

Turning now to epidemics of a more restricted character, amongst which may be mentioned an outbreak that occurred in the Mediterranean squadron, as recorded by *Bryson* (15), where in each ship of the squadron pleuropneumonia occurred, of a severe and complicated type; we find mention by *Mendelsohn* of an epidemic, in 1867, at the Akershus prison at Christiania, reported by *Dahl* (16). In the first five months of the year 62 out of 360 prisoners were attacked with pneumonia, of whom five died. Six nurses were also attacked, and the outbreak was referred to the bad ventilation of the dormitories.

*Gründler* (17) published details of an epidemic in the Magdeburg garrison, that lasted from September, 1873, to June, 1874; the number of cases of pneumonia amongst the civil population during this period not being above the normal. The garrison numbered 5616 men; and of these 73 were taken ill, particularly recruits, and those of an infantry regiment occupying barracks erected on a marshy soil and badly ventilated. Four cases were fatal, *i.e.*, 5·48 per cent., so that the outbreak was not a severe one. The winter was a mild one, but the men were subjected in their morning and evening drills to changes of temperature, which may also, it is thought, have had some effect in determining the attack.

The most circumstantial account of this class of pneumonia epidemics is that given by *Adolf Kühn* (18), as occurring in the Moringer Reformatory in 1874-5. In 1874 there was a great increase in the number of inmates, so that not only was the cubic space of the dormitories much diminished, but beds had to be placed in the corridors. The building was then enlarged to accommodate 237, but the numbers increased to 351; and as a consequence there was, during 1874, a great increase in all forms of disease of the respiratory organs, pneumonia and phthisis especially. Amongst them were eleven cases of lobar pneumonia of a peculiar type, and running a course more re-



sembling that of an infectious disease. At the same time there were 43 cases of gastro-intestinal disorder of a typhoid character associated with bronchitis and lobular pneumonia. At the end of 1874 the buildings were again extended to accommodate 414 inmates, but even this extension failed to keep pace with the number admitted, so that the effects of over-crowding were still felt. During 1875, there were 83 cases of pneumonia, 45 of which were of a severe type. Several of the cases were preceded by malaise, lasting a few days; then occurred a rigor with rise of temperature to  $104^{\circ}$  or over, but with very scanty physical signs of pneumonia. In such cases there were also swelling of the spleen, albuminuria, and in many diarrhœa. In others the signs of pneumonia were manifest by the third or fourth day, and the disease assumed the "wandering" type. All the cases were complicated with pleurisy (four with empyema), and in one-fourth of the whole number there was pericarditis. Many had cerebral symptoms. Coincident with these severe and well-marked cases, were others of slight illness, mostly recorded as catarrh, but which from their general similarity were regarded as abortive forms of the graver disease. Post-mortem examinations were made in 16 cases; meningitis was present in four out of seven cases in which the head was examined; pericarditis was found in 10 cases, an enlarged spleen in 13, and some swelling of the intestinal follicular glands in many. Kühn thinks there can be little question of the "typhoid" character of this epidemic, and that it was apparently due to the over-crowding.

Another prison endemic is mentioned by *Rodman* (19) as occurring in the Kentucky State Prison at Frankfort, Ky., in February, 1875. Pneumonia had been unusually prevalent in the country during the autumn and winter, but cases had ceased to occur when the remarkable outburst of disease in the prison commenced on February 24th. The prison cells, 648 in number, were greatly overcrowded in that month, 694 prisoners being lodged on February 1st, and 735 on March 1st. During 1874 there had been 75 cases of pneumonia in the prison, with a mortality of eight per cent.; and from January 1st, 1875, to February 24th 16 cases, with one death. From February 24th to July 1st, there were 118 cases, and 98 of these were of a specially

asthenic type, with symptoms pointing to a specific poisoning. Twenty-five died, and of these 14 were new prisoners. The sanitary condition of the prison was very bad, for owing to the uncleanly habits of the inmates, the cells and corridors, especially of the upper floors, reeked with fœtid emanations. The outbreak belongs to the class of "pythogenic" pneumonia without a doubt; and the enormous mortality (26 per cent.), justifies the condemnation passed on the prison buildings.

*Kerschensteiner* (20) describes an epidemic which occurred in the male prison at Amberg, and which might also be attributed to the insanitary condition of the dormitories, but the cases were more strictly on the classical type of pneumonia than those occurring at Moringen. From January 1st to May 28, 1880, 161 inmates were attacked, of whom 46 died, or 28·5 per cent. The epidemic was thus a very fatal one, the cases being of the asthenic type. *Kerschensteiner* was inclined to regard it as due to some special miasm.

*Knoevagel* (21) has record an outbreak which took place at the garrison at Cologne, from October 1st, 1879, to May, 1880. During this period, amongst 389 patients admitted into hospital, there were 80 cases of pneumonia, seven of which proved fatal. Most of the cases came from barracks, which were ill-ventilated and noisome; and, as in other instances, the recruits suffered most.

The question of contagion, or the transmission of the disease from one individual to another, has been exemplified in the history of many of the foregoing epidemics. The alternative explanation of the existence of some local condition of the dwellings, &c., is in many cases quite as worthy of credence. But in some of the following instances there is very little room for escape from the first-named hypothesis.

Writing in 1876 to a Paris journal, *Dr. H. J. Hardwick* (22), of Sheffield, endeavours to awaken an interest in France on the subject of epidemic pneumonia, which at that time was not much accepted in England. He mentions the case of a minister suffering from acute pneumonia, and attended in his illness by a relative, who not only fell ill of the same disease, but who apparently communicated it to another relative. In another case some relatives, who visited an old man dying from pneumonia,

were each attacked by the same disease. He also alludes to a small village epidemic, in which six persons were successively attacked, in 1873, the neighbour of the first case being taken ill a few days after him. Dr. Hardwick is clearly of opinion that pneumonia may occur under two forms,—that due to exposure to cold, and that to zymotic influences.

One of the most striking instances of pneumonia spreading through a household is given by *Adolph Müller* (23). It occurred at Ganzenhausen in the winter of 1873. The first to fall ill was the mother (*a*) æt. 58, on November 25th; on December 6th the father (*b*) æt. 64; on December 7th the son (*c*) æt. 18, and about the same date a daughter (*d*), who did not live with her parents, but who had been attending on her mother during her illness; and a grandchild (*e*), 5 years of age, took ill on the 10th of December. In all of the cases but (*e*) there was a marked initial rigor; and in all but (*a*) the attack was accompanied with gastric symptoms; (*c*) and (*d*) suffering severely from vomiting. There was great prostration in all, the parents (*a*) and (*b*) being also delirious, and having an irregular feeble pulse. The physical signs of pneumonia were marked, and the expectoration characteristic. The inflammation involved the left upper lobe in (*a*) and (*e*); the right upper lobe in (*b*) and the right lower lobe in (*c*). All recovered by crisis, which took place on the 9th, 7th and 5th days respectively.

In the Wagner Clinic at Leipsic, in the summer of 1880, *Heitzel* (24) met with the cases of three sisters, aged 7, 9 and 11 years, who fell ill with croupous pneumonia consecutively. In each case the disease ran a typical course, and terminated favourably. There had been no exposure to cold; but it is interesting, as suggestive of a family tendency, to note that the eldest sister, who was the first to be attacked, had three times previously suffered from pneumonia.

*Ritter* (25) gives an account of an endemic of pneumonia in the house of a relative. Within a period of five days, March 13 to 18, 1879, five persons dwelling in the same house fell ill, and two others, who had often visited at the house, were later attacked. Of these seven cases three proved fatal, the disease being of a typhoid type, with splenic swelling, &c.



Mendelsohn mentions that *Bielski* (26) records the incidence of pneumonia upon nine out of ten inhabitants of one house. They were attacked within two weeks of each other, and all recovered. Eight out of the nine cases involved the right lung. In this case there was not only overcrowding, but the house lay in the vicinity of a graveyard.

This review by no means exhausts all that has been written upon the subject,\* although it embraces the majority of the records of actual epidemics. Upon the general question of infectious pneumonia there have been writings too numerous to mention. Amongst those who have written most forcibly in support of pneumonia being under all circumstances a specific and constitutional fever, may be cited *Austin Flint* (27), *Juergensen* (28), *Könhorn* (29), and *Mendelsohn* (3). Könhorn supports his contention by an appeal to his experience of eight years' of the disease amongst the Wesel garrison; and he shows how some barracks yielded a far larger proportion of pneumonia than others. He also believes that relapsing fever is etiologically connected with pneumonia. Mendelsohn—as before stated—reviews the whole literature and supplies some remarkable observations of his own in the second medical clinic at Berlin, which are worthy of citation. One of these was the case of a coachman, 35 years of age, who with his family (wife, and three children from 1 to 5 years old) moved on April 1, 1883, to a new lodging, consisting of one room and a kitchen. The lodging had been left by its former tenants in a state of horrible filth, and for some days the man and his wife were occupied in cleansing it. On April 20 the woman was attacked with pneumonia; next day the eldest child, and the following day the youngest. On the 26th the father was also attacked. Both the children were removed to hospital, where they contracted measles and scarlet fever, and died. The parents passed through a severe attack of pneumonia; in the woman's case, complicated with otitis. They had never been ill before and attributed (with reason) their attack to the unwholesome state of the house. The man on returning home at the end of May had a recurrence of the disease and was again

\* The pathological aspect of the subject has been omitted here, including the recent observations of Friedländer and others.

admitted into hospital. The other observation refers to pneumonic contagion, and was made in the same clinic. A patient in the remitting stage of typhoid fever was transferred from the bed he had occupied during the attack to one on the opposite side of the ward. In a few days he became feverish, and dulness was made out over the lower lobe of the right lung. He died eight days afterwards, and it was found that the typhoid ulcers were healed, but that the lung was hepatised in the above-named region. On inquiry, it appeared that the bed to which this patient had been transferred had just before been occupied by a very severe case of pneumonia, then convalescent; and that the mattress had not been changed. In another instance, a typhoid patient was admitted into a ward which contained many cases of pneumonia. The fever at first took a mild course, but in a few days pneumonia developed, and death took place on the 17th day. There was found marked hepatisation of the right lower lobe, not the hypostatic consolidation peculiar to fever. Mendelsohn remarks, that pneumonia following typhoid has not often been observed in the clinic, and believes that, in each of these instances, the pneumonia must have been acquired in the ward, possibly by direct infection.

Amongst other contributors to the general subject are *Lépine* (30), *Demmler* (31), *Bonnemaison* (32), who gives instances of epidemics at Toulouse, mostly associated with influenza, an association noted by *Wilson Fox* (33) and others, but obviously not sufficient to account for all instances of epidemic pneumonia.

Lastly. The questions whether all pneumonia is zymotic or at any rate, constitutional, as *Flint* and *Juergensen* hold, or whether there exists a zymotic form and a simple form due to exposure to cold, as *Hardwick* believes; or whether the evidence in support of a specific theory is unsubstantial, as *Hallopeau* (34) considers, and that the relation of pneumonia to meteorological and climatic conditions is more exact than many admit, as *Seibert* (35) has elaborately shown, cannot be discussed in this place. There can be no question as to the existence of epidemics of pneumonia, but this fact is only one argument out of many that seem to point in the direction to which pathology and clinical observation are tending.

SIDNEY COUPLAND.

BIBLIOGRAPHY.

1. HIRSCH, A.—'Handbuch der historisch-geographischer Pathologic.' 1862-64. Vol. ii., p. 38, *et seq.*
2. HAESER, H.—'Lehrbuch der Geschichte der Medicin.' 3rd ed. 1882. Vol. iii., p. 379.
3. MENDELSON, M.—Die Infectiöse Natur der Pneumonie. 'Zeitschrift für Klinische Medicin.' 7 btr Bd. 1883. p. 178.
4. SANDERS, E.—Croupous Pneumonia, an acute infectious disease. 'Seguin's Archives of Medicine.' June and August, 1881. This article comprises a full bibliography.
5. HJALTELIN, J. H.—Epidemic Pneumonia in Iceland in the year 1863. 'Edinburgh Medical Journal.' May, 1864.
6. BANTI, G.—De la Pneumonie Infectieuse, trad par E. Vaisson. 'Archives générales de Medicine.' 1880. ii., p. 36.
7. MOELLMANN.—Zur Ätiologie der croupösen Pneumonie. 'Berliner Klinische Wochenschrift.' 1879. No. 14, p. 155.
8. FELDHAUSEN, J.—Einige Mittheilungen über die vom 1 April 1877 bis 1. April, 1879, in der Med. Klinik in Göttingen beobachteten Pneumonien. Göttingen, 1879. Abstract in 'Schmidt's Jahrbuch,' 195. 1882. p. 256.
9. BUTRY.—Eine maligne Pneumonie-epidemic. 'Deutsch. Archiv. f. Klin. Med.' 1881. xxix., p. 193.
10. v. HOLWEDE UND MÜNNICH.—Endemisches Auftreten von croupöser Pneumonie, briefly abstracted in 'Berliner Klin. Wochenschrift.' 1881. No. 23, p. 332 and elsewhere.
11. PENKERT.—Pneumonia crouposa epidemica. 'Berliner Klin. Wochenschrift.' 1881. Nos. 40 and 41, p. 577.
12. SCHEEF, J.—Ueber eine Pneumonie-Epidemie, Croupöse Pneumonie-Beobachtungen aus der Tübingen Poliklinik; herausgegeben von. Th. Jürgensen. Tübingen: 1883. p. 97.
13. SCHMID, H.—Ueber ein epidemisches auftreten von Pneumonia crouposa. 'Berliner Klin. Wochenschrift.' 1883. No. 23, p. 346.
14. SENFFT, A.—Beitrag zur epidemischen Pneumonie. 'Berliner Klin. Wochenschrift.' 1883. No. 38, p. 580.
15. BRYSON.—Epidemic of pleuropneumonia in some ships of the Mediterranean fleet. 'Med. Times and Gazette,' January, 1864. Quoted in (3) and (4).
16. DAHL.—'Dublin Journal of Med. Sci.' 1875.
17. GRÜNDLER.—Statistische Mittheilungen über die Erkrankungen von Lungenentzündung in der Garnison Magdeburg. 'Deutsch. militär-ärztl. Zeitschrift' iv., 2, p. 59. 1875. Abstr. in 'Schmidt's Jahrbuch,' 195. 1882. p. 254.
18. KÜHN, A.—Die contagiöse Pneumonia. 'Deutsch. Arch. f. Klin. Med.' 1878. xxi., p. 348, and Zur Ätiologie u pathologische Anatomie endemischer Pneumonie. 'Berliner Klin. Wochensch.' 1879. No. 37, p. 552.
19. RODMAN, W. B.—Endemic of Pythogenic or Miasmatic-infectious Pneumonia. 'American Journal of Med. Sci.' 1876, Jan. p. 76.
20. KERSCHENSTEINER, J.—Ueber infectiöse Pneumonie. 'Bayr. ärztl. Intellig. Bl.,' xxviii., 20. 1881. Abstract in 'Schmidt's Jahrbuch.' 1882. 195, p. 256.
21. KNOEVAGEL.—'Beiträge zur statistik und Ätiologie der Lungenentzündungen beim Militär.' 'Deutsch. mil. ärzl. Ztsch.,' xi. 1882. Abstract in 'Schmidt's Jahrbuch.' 1882. 165, p. 254.
22. HARDWICK, H. J.—Pneumonie, maladic infectieuse, zymotique et contagieuse. 'Gazette Médicale de Paris.' 1876. No. 43, p. 515.
23. MÜLLER, A.—Endemische Pneumonie. 'Deutsch. Archiv f. Klin. Med.' 1878. xxi., p. 127.
24. HEITZEL.—Ueber infectiöse Pneumonie. Leipzig. 1883. Quoted by (3) and abstract in 'Börner's Jahrbuch.' 1884. p. 334.
25. RITTER, J.—Beitrag zur Frage des Pneumotyphus. 'Archiv f. Klin. Med.' 1880. xxv., p. 53. Quoted by (3).
26. BIELENSKI.—In 'Medycyna.' 1882. 13. Quoted by (3).



27. FLINT, A.—Pneumonic fever. 'Trans. of Med. Soc. of the State of New York.' 1877. Also 'Treatise on the Principles and Practice of Medicine.' 5th ed., p. 160.
28. JUERGENSEN.—In 'Ziemnssen's Cyclopædia.' Amer. transl. Vol. v.—Croupöse Pneumonie. Tübingen. 1883.—Ueber genuine Pneumonie. 'Vortrag un Dritter Congres für Innere Medicin.' Berlin. April, 1884. 'Berl. Klin. Wochenschr.' 1884. No. 17, p. 270.
29. KÖHNHORN.—Zur Ätiologie der Lungenentzündung. 'Vjhrschr. f. Ger. Med.' Juli, 1881. Abstract (full) in 'Schmidt's Jahrbuch.' 1882. 195, p. 250.
30. LÉPINE, R.—Art. 'Pneumonie,' in 'Nouv. Dict. de Med. et de Chir. Pract.' xxviii. 1880.
31. DEMMLER, H.—Etude sur les Pneumonies infectieuses. 'Th. de Paris.' 1882. Abstract in 'Archives Gén. de Méd.' 1883. i., p. 246.
32. BONNEMAISON.—Mémoire sur les Pneumonies malignes. 'Memoires de la Societé des Hôpitaux de Paris.' 1875. Vol. xii.
33. WILSON FOX.—Article 'Pneumonia' in 'Reynolds's Syst. of Medicine.' Vol. 3. 1871.
34. HALLOPEAU, H.—La doctrine de la Fièvre Pneumonique. 'Revue des Sciences Medicales.' 1878. xii., p. 730.
35. SEIBERT, A.—The influence of meteorological conditions upon the causation of Croupous Pneumonia. 'Amer. Journ. of Med. Sci.' Jan. 1882. p. 108. Witterung und croupöse Pneumonie. 'Berl. Klin. Wochenschr.' 1884. Nos. 18 and 19.

For references to literature on the occurrence of micro-organisms in the inflammatory exudation and sputa of pneumonic patients, see Mendelsohn's article (3), and article by E. Bricon in 'Le Progrès Médical,' December 8 and 15, 1884, also the recent debate in the German Medical Congress, 'Deutsch Med. Woch.,' 1884, Nos. 17 and 18.

## PART II.

## REPORT ON THE RETURNS.

*Number and Distribution of Cases.*

IN the first number of the *Record* a preliminary report upon acute pneumonia, containing an analysis of 350 returns, was published, and the hope was then expressed that when thrice that number had been collected, an opportunity would be given for drawing wider and juster inferences upon many points. We have now before us the returns of 1065 cases, which are tabulated in the appendix (No. 717 being omitted), and for the sake of convenience this number has been mainly dealt with in three series, containing nearly approximate numbers. Thus—

Series A includes Nos. 1 to 350.

„ B „ „ 351 to 700.

„ C „ „ 701 to 1066 (less No. 717).

Of these cases we find the returns in about 14 to be very defective. These are contained in series C and are partly compensated by the larger number included in that series. So that to all intents and purposes, the three series above named may be fairly contrasted with one another on many points. Again, 24 other returns are defective, wanting details on several heads; and about 50 are defective in some details. The precise number of cases which are thus lacking in particulars will be found enumerated under each heading in the following abstracts.

The *period actually covered* by these returns is an extensive one, far exceeding the time during which collective investigation has been in progress. This is explained by the fact that many returns have been made of cases under observation prior to the date at which the report was sent in.

No dates are affixed to 16 of these reports (Nos. 517, 700, and Nos. 828 to 841 incl.).

The following table contains a numerical analysis of the returns in each series—from May, 1882, to April, 1884. Prior to May, 1882, there occur 55 cases, distributed in various months. These may be summarised, with respect to the years, thus:—

1876 . . . . .	1 case.	Deaths 0.
1877 . . . . .	1 „	„ 0.
1878 . . . . .	3 cases.	„ 2.
1879 . . . . .	6 „	„ 2.
1880 . . . . .	12 „	„ 0.
1881 . . . . .	10 „	„ 0.
Jan.-April, 1882 . . . . .	22 „	„ 3.

	Series A.		Series B.		Series C.		Total.		
	Rec.	Died.	Rec.	Died.	Rec.	Died.	Rec.	Died.	
Not dated . . . . .	—	—	2	—	12	2	14	2	16
Prior to May 1882 . . . . .	29	5	10	1	9	1	48	7	55
May 1882 . . . . .	10	2	—	1	1	—	11	3	14
June „ . . . . .	19	1	—	—	1	—	20	1	21
July „ . . . . .	15	3	—	—	2	—	17	3	20
Aug. „ . . . . .	2	—	—	—	—	—	2	—	2
Sept. „ . . . . .	6	3	—	—	—	—	6	3	9
Oct. „ . . . . .	13	3	3	—	—	—	16	3	19
Nov. „ . . . . .	24	7	1	1	2	1	27	9	36
Dec. „ . . . . .	46	6	5	1	2	—	53	7	60
Jan. 1883 . . . . .	18	10	5	1	—	—	23	11	34
Feb. „ . . . . .	31	5	16	4	3	—	50	9	59
March „ . . . . .	27	12	17	2	1	1	45	15	60
April „ . . . . .	36	10	35	5	5	1	76	16	92
May „ . . . . .	5	2	62	11	5	1	72	14	86
June „ . . . . .	—	—	44	10	6	5	50	15	65
July „ . . . . .	—	—	14	6	10	1	24	7	31
Aug. „ . . . . .	—	—	21	5	4	1	25	6	31
Sept. „ . . . . .	—	—	9	3	2	3	11	6	17
Oct. „ . . . . .	—	—	19	6	9	—	28	6	34
Nov. „ . . . . .	—	—	28	1	41	9	69	10	79
Dec. „ . . . . .	—	—	1	—	52	12	53	12	65
Jan. 1884 . . . . .	—	—	—	—	43	12	43	12	55
Feb. „ . . . . .	—	—	—	—	22	4	22	4	26
March „ . . . . .	—	—	—	—	43	11	43	11	54
April „ . . . . .	—	—	—	—	25	—	25	—	25
	281	69	292	58	300	65	873	192	1065

Although an inquiry that deals with but a small proportion of the total number of cases occurring in the country is hardly one from which to draw precisely accurate conclusions with respect to the *seasonal* prevalence of the disease, yet it may be taken for granted that the number of returns made to us within a given period do bear some relation to that prevalence. We have no means of ascertaining the actual amount of pneumonia in the United Kingdom, but must be content with such indications as these returns afford. Without then laying undue stress upon these results we have collated the cases occurring in the different seasons for the past two years, and compared their mortality. From this it will appear that the largest number of



returns was made in spring, 1883, and that in these cases the highest mortality occurred in autumn, 1882, and summer, 1883.

		Cases.	Deaths.	Mortality.
1882	spring (incomplete), (May) ... ..	14	3	
"	summer (June, July, August) ... ..	43	4	9·3 per cent.
"	autumn (September, October, November) ... ..	64	15	23·4 "
1882-3	winter (December, January, February)... ..	153	27	17·6 "
1883	spring (March, April, May) ... ..	238	45	18·8 "
"	summer (June, July, August) ... ..	127	28	22 "
"	autumn (September, October, November) ... ..	130	22	16·9 "
1883-4	winter (December, January, February)... ..	146	28	19·1 "
1884	spring (incomplete), (March, April) ... ..	79	11	
		994	183	18·4 "

### *Observers and Localities.*

Concerning the geographical distribution of the disease no inferences can be drawn from these returns, forasmuch as the numbers indicate rather the relative activity of the collectors than the comparative prevalence of the disease.

The total number of reporters of cases analysed in the present report amount to 482.

29 cases were reported on by 1 observer...	...	...	...	29
14 " " " " 1 " " " " " " " " " "	1	"	"	14
12 " " " " 3 " " " " " " " " " "	3	"	"	36
10 " " " " 3 " " " " " " " " " "	3	"	"	30
9 " " " " 3 " " " " " " " " " "	3	"	"	27
8 " " " " 5 " " " " " " " " " "	5	"	"	40
7 " " " " 8 " " " " " " " " " "	8	"	"	56
6 " " " " 8 " " " " " " " " " "	8	"	"	48
5 " " " " 15 " " " " " " " " " "	15	"	"	75
4 " " " " 29 " " " " " " " " " "	29	"	"	116
3 " " " " 40 " " " " " " " " " "	40	"	"	120
2 " " " " 108 " " " " " " " " " "	108	"	"	216
1 case was " " 258 " " " " " " " " " "	258	"	"	258
—482				—1065

The *localities* from which these cases are reported are too numerous to be given in detail. Of 1054 cases in which the locality is indicated, 826 are reported from England, 87 from Wales, 99 from Scotland, 34 from Ireland. The remainder from places outside the United Kingdom, viz.:—Cairo, 1; Delhi, 2; Monte Video, 1; Rome, 2; and Sydney, 2.

#### *England (826).*

Berkshire .....	7	Gloucestershire .....	15	Norfolk .....	8	Warwickshire..	22
Cambridgeshire...	11	Hants.....	16	Northampton....	6	Wilts.....	1
Cheshire.....	22	Hereford .....	2	Northumberland..	4	Worcester-	
Cornwall .....	6	Herts .....	23	Notts .....	10	shire .....	30
Cumberland .....	7	Hunts.....	3	Shropshire .....	36	Yorkshire .....	72
Derbyshire.....	11	Kent ... ..	76	Somerset .....	21		
Devon .....	7	Lancashire .....	184	Suffolk .....	4	Isle of Wight...	11
Dorset .....	16	Lincolnshire .....	17	Staffordshire .....	42	Channel	
Durham.....	8	Middlesex,includ-		Surrey .....	56	Islands .....	5
Essex .....	3	ing London ...	54	Sussex .....	10		

#### *Wales (87).*

Anglesea .....	7	Flint .....	3	Monmouth-		Montgomery-	
Carmarvon.....	3	Glamorgan.....	54	shire .....	8	shire .....	1
Denbigh.....	6	Merionethshire ...	5				

*Scotland (99).*

Aberdeenshire ...14	Dumfries ..... 1	Inverness ..... 1	Perthshire ..... 1
Ayr..... 9	Edinburghshire.. 11	Kincardine ..... 5	Renfrew.....11
Banff ..... 2	Fifeshire ..... 8	Lanarkshire ..... 6	Ross and Cro-
Clackmannan ... 2	Forfar.....10	Linlithgowshire... 1	marty ..... 6
Dumbarton ..... 2	Haddington ..... 9		

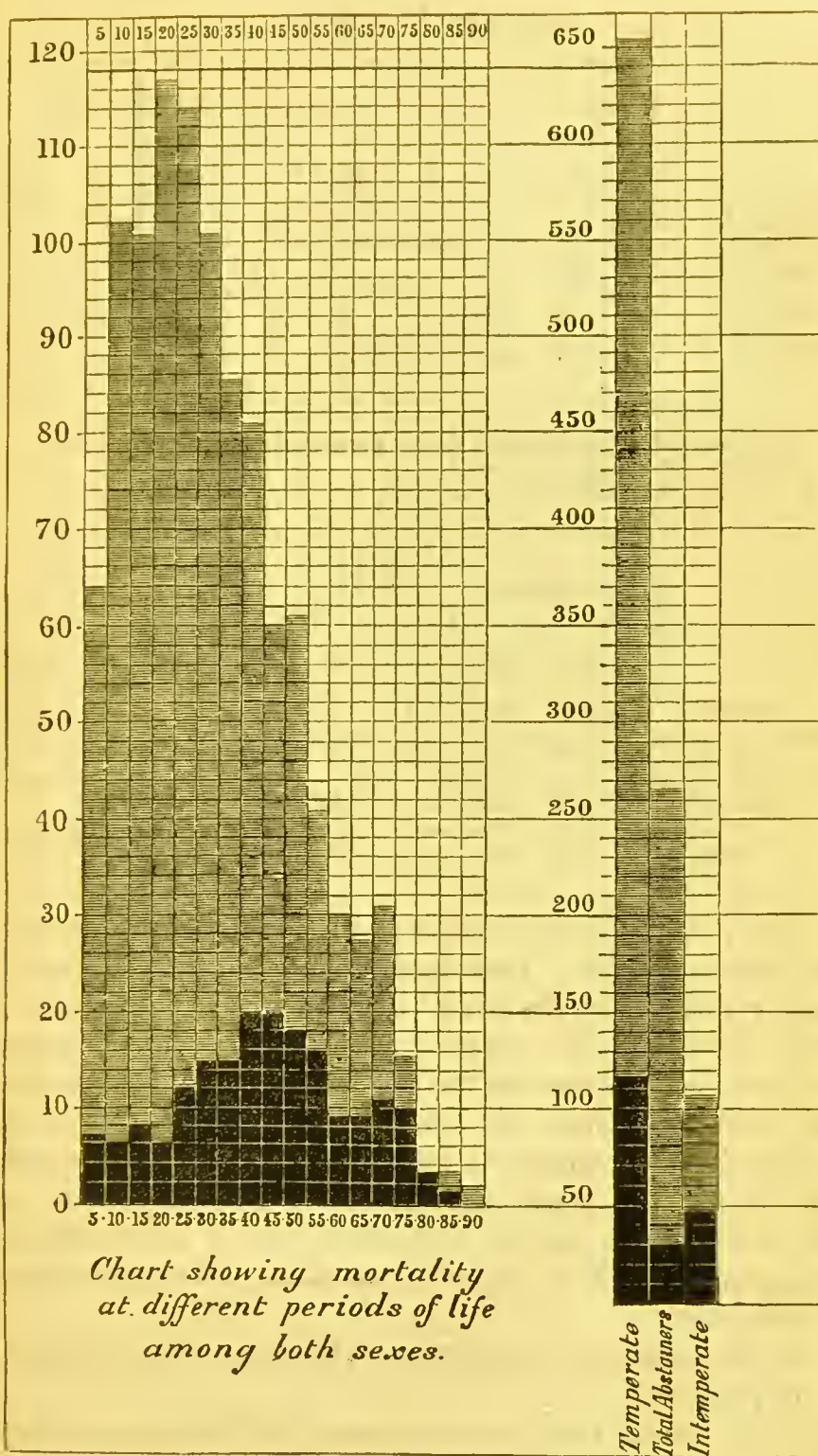
*Ireland (34).*

Antrim ..... 3	Dublin Co.....12	Londonderry..... 4	Tyrone ..... 1
Armagh ..... 1	Kerry..... 1	Limerick ..... 1	Waterford... 2
Donegal..... 2	Kilkenny ..... 7		

*Abstract I.—Sex, age, &c.*

	Males.			Females.			Totals.			Mortality.
	Rec.	Died.	Total.	Rec.	Died.	Total.	Rec.	Died.	Total.	
— 5 years	34	3	37	23	4	27	57	7	64	10·9 p. c.
6-10 "	64	6	70	32	—	32	96	6	102	5·9 "
11-15 "	58	2	60	35	6	41	93	8	101	7·9 "
16-20 "	80	4	84	31	2	33	111	6	117	5·1 "
21-25 "	72	7	79	30	5	35	102	12	114	10·5 "
26-30 "	56	8	64	30	7	37	86	15	101	14·8 "
31-35 "	46	10	56	24	5	29	70	15	85	17·6 "
36-40 "	41	13	54	20	7	27	61	20	81	24·7 "
41-45 "	35	17	52	5	3	8	40	20	60	33·3 "
46-50 "	24	14	38	19	4	23	43	18	61	29·5 "
51-55 "	17	9	26	8	7	15	25	16	41	39·5 "
56-60 "	14	7	21	7	2	9	21	9	30	30· "
61-65 "	11	7	18	8	2	10	19	9	28	32·1 "
66-70 "	13	4	17	7	7	14	20	11	31	35·5 "
71-75 "	1	6	7	4	4	8	5	10	15	60·8 "
76-80 "	—	—	—	—	3	3	—	3	3	
81-85 "	2	—	2	—	1	1	2	1	3	
86-90 "	2	—	2	—	—	—	2	—	2	
Age not stated.	14	3	17	2	2	4	16	5	21	
	584	120	704	285	71	356	869	191	1060	

The sex and age are stated in 1039 cases, the sex not being stated in five (four recoveries and one death), and the age not given in 21 (16 recoveries and five deaths). In the foregoing table the number of each sex within periods of five years, and the mortality in the same period are stated. From this it appears that amongst the males there were 704 cases and 120 deaths, mortality 14·2 per cent.; and of females, 356 cases and 71 deaths, mortality 19·9 per cent. Further, that the maximum mortality of both sexes below the age of 70 is to be found between 50 and 55 years, viz.: 39·5 per cent.; the minimum mortality being between five and ten years, viz.: 5·9 per cent. (*vide* Chart). This result is in harmony with the results obtained by Dr. Longstaff.





*Abstract II.—Habits.*

	Temperate.		Total Abstainers.		Intemperate.		Not stated.		Total	
	Rec.	Died.	Rec.	Died.	Rec.	Died.	Rec.	Died.	Rec.	Died.
Series A.	178	43	72	10	21	15	10	1	281	69
Series B.	177	35*†	88	8‡	19	11	8	4	292	58
Series C.	186	36	79	10	20	19	15	—	300	65
Total.	541	114	239	28	60	45	33	5	873	192

\* No. 399.—Died from apoplexy during convalescence from pneumonia.

† No. 591.—Formerly intemperate.

‡ No. 520.—Total abstainer for three months; previously intemperate.

	Food Sufficient.		Food Insufficient.		Not stated.		Total.	
	Rec.	Died.	Rec.	Died.	Rec.	Died.	Rec.	Died.
Series A.	256	62	22	7	3	—	281	69
Series B.	279	52	12	6	1	—	292	58
Series C.	278	61	11	2	11	2	300	65
Total.	813	175	45	15	15	2	873	192

Under this head are considered the replies to the questions as to food and drink. Considerable allowance must be made in these respects, since the terms “temperate” and “sufficiency of food” represent only a relative and not an absolute statement. This fact may account for the not inconsiderable divergence in the mortality between the temperate and the total abstainers, so that the latter appear to even greater advantage than they are entitled to. Again, amongst the “total abstainers” are included a larger number of children than of adults, and the mortality rate with the former is, as above stated, much lower. (*Vide Chart.*)

Of 655 returned as “temperate” 114 died, giving a mortality of 17·4 per cent.

Of 267 returned as “total abstainers” 28 died, giving a mortality of 10·4 per cent.

Of 105 returned as "intemperate" 45 died, giving a mortality of 42·8 per cent.

And as to food—

Of 988 stated to have "sufficient food" 175 died, or 17·7 per cent.

Of 60 stated to have "insufficient food" 15 died, or 25· per cent.

The average mortality of the whole number (192 deaths amongst 1065 cases) amounts to 18 per cent.

*Abstract III.—Illnesses concurrent with Pneumonia.\**

	Pneu- monia.		Bronch		Tonsil.		Erysip.		Occurring in District.								In House.		
									Scarlat.	Typhoid.	Measles.	Herpes.	Diphtheria	Rh. Fever	Rötheln.	Mumps.	"Fever."	Herpes.	Typhoid†
	H.	D.	H.	D.	H.	D.	H.	D.											
Series A....	20	120	35	140	20	94		70	46	28	23	16	6	5	3			1	1
Series B....	70	151	29	149	9	102	3	55	28	31	20	38	4	5	3	3	3	5	1
Series C....	25	130	31	150	16	116	2	63	54	32	16	30	9	1	2	1	2	4	3
	115	401	95	439	45	312	5	188	128	91	59	84	19	11	8	4	5	10	5

The above enumeration refers to 317 cases of Series A., 32 wanting.

" " " 315 " " B., 35 "

" " " 327 " " C., 39 "

No disease occurs in the same house with pneumonia so frequently as pneumonia itself. This coincidence of two or more cases of pneumonia in the same house is to be found in the proportion of 1 to 8·3.

The only other diseases apt to concur with pneumonia in the same house are bronchitis and tonsillitis. Enteric fever is met with in the same house in but five instances, and in three of these typhoid is in the district as well.

The occurrence of other cases of pneumonia (one or more) in the same house with the case reported is met with in 70

\* The precise accuracy of these figures cannot be asserted. There is sometimes doubt as to number of districts reported from by one observer. The errors, however, are insignificant, and cannot interfere with any of the general conclusions to be derived from the table.

† These cases of typhoid in the same house are Nos. 147, 488, 738, 852. and 870.

instances in Series B., or a fifth of the entire number of the series, very largely exceeding the number of examples of a like occurrence in either of the other series, or in the two put together.

The prevalence of bronchitis in the same house with pneumonia is not in harmony with the prevalence of pneumonia itself in the same house. Thus bronchitis is in the same house with pneumonia in 35 instances of the first series (that of the least prevalence of pneumonia in the same relation) while it is in the same house with it in but 29 instances of the second series (that of the greatest prevalence of pneumonia in the same relation).

The prevalence of bronchitis in the same *district* with the reported cases of pneumonia does not widely differ for the three series. Such differences as appear are quite out of accord with the corresponding numbers for pneumonia.\*

The frequent occurrence of erysipelas in the same district with pneumonia is noteworthy, but here again there is no correspondence in relative frequency, and the occurrence of erysipelas in the same house with pneumonia is extremely rare. The only affection at all parallel with pneumonia in respect of frequency is herpes.

Of the specific fevers it is only necessary to observe that there is no intimate or constant connection between any of these and pneumonia. Enteric fever occurs in the same district pretty equally throughout the three series, that is to say in about an eleventh of the whole number of districts.

The rarity of diphtheria is noticeable. It is met with in the same house with pneumonia in but one instance.

The series (B.) of greatest prevalence of pneumonia is also the series of least fatality. On particular pages of the Returns great frequency will be found to concur with a low rate of mortality. Thus from 412 to 485 (pp. xliv.—li.) a period of great frequency,

\* It will be found, speaking generally, that while the maximum prevalence pneumonia does not always coincide with maximum prevalence of bronchitis (*vide* pp. xciv.-v. App.) the minimum prevalence of the two very nearly correspond (*vide* pp. xxv.-vi. App.).

Much the same may be said for tonsillitis and erysipelas. The occurrence of tonsillitis in the same house with pneumonia (like the occurrence of bronchitis) is greatest for Series A., least for Series B.



there are but three deaths for 74 cases, notwithstanding that the sanitary conditions are worse than usual. On the other hand, with lessened frequency of pneumonia, as for example on pp. xviii. —xxi., or pp. lxxxiii., xciv., xcv., there are respectively 36 cases with 10 fatal and 29 cases with 6 fatal.

*Abstract IV.—Sanitary States of Houses and Districts respectively, whence Cases of Pneumonia are reported.*

	Sanitary Condition of Houses reported from.				Sanitary Condition of Districts reported from				Cases reported "Good," both House and District.	Cases reported "Indiff." or "Bad," both House and District.	Cases reported "Bad," both House and District.
	Good.	Indiff.	Bad.	Doubtful.	Good.	Indiff.	Bad.	Doubtful.			
Series A...	172	135	35	8	168	135	33	14	115 (26 fatal)	114 (23 fatal)	15 (3 fatal)
Series B...	185	120	40	5	178	142	19	11	144 (26 fatal)	126 (16 fatal)	10 (2 fatal)
Series C...	202	111	25	27	221	96	22	26	176 (37 fatal)	81 (12 fatal)	7 (1 fatal)
	559	366	100	40	567	373	74	51	435 (89 fatal)	321 (51 fatal)	32 (6 fatal)

The proportion of cases in which the sanitary condition of the houses reported of is good to the number in which it is reported as "bad" or "indifferent" is about 1·2 to 1: of the districts about 1·3 to 1.

Reckoning by *houses* only, the sanitary condition of Series C. is the best, and of series A. the worst. If, however, the instances where sanitation is reported "good," both for house and district be compared with those in which it is "indifferent" or "bad" for both house and district (as has been done in the table above) it will be found that Series B. shows the worst of the three. Yet this is the series of lowest mortality; and the 126 exhibiting bad sanitary conditions have a mortality of but 16, or 1 in 8; while the 144 under good sanitary conditions have a mortality of 26, or 1 in 5½.

Furthermore, if certain exceptional cases of the worst sanitary

conditions be selected we get 32, with a mortality of six, one of these being an infant insufficiently fed, and one a man of 72.\*

No connection could be discovered between insanitary conditions and any particular seat, duration or mode of termination of pneumonia.

*Abstract V.—Locality and Climatic Conditions.*

With the object of ascertaining whether any particular condition of weather and locality exerts a preponderating influence in regard to the prevalence of pneumonia, the card of inquiry contained references to a few of the more salient points in these respects. In order to simplify the question as much as possible, without depriving the answers of importance, the statements required were made in very general terms, nor does the result fall to be instructive.

First, as to the *locality* of the patient's residence. Observers were asked to state whether or not it was on (*a*) high or low ground, on (*b*) dry or damp soil, and (*c*) in an exposed or confined situation. It could hardly be expected that a categorical affirmative or negative could be given in every case to such questions; and hence it is not surprising that by a certain number the questions should have remained unanswered, or that occasionally qualifying expressions should have been used. Replies to one or more of the questions have been made by 1032 observers, with the following results:—

(*a.*) Amongst 957 returns, 565, or 59 per cent., state that the pneumonia occurred in localities seated on *high* ground, and 392, or 41 per cent., on *low* ground.

(*b.*) Amongst 885 returns, 486, or nearly 55 per cent., state the nature of the soil to be *dry*, and 399, or 45 per cent., to be *damp*.

\* The influence of insanitary conditions in originating pneumonia or favouring its spread should be seen, if anywhere, in Series B., where we have 70 examples of multiple pneumonia (*i.e.*, two or more examples in the same house). These houses are reported as "good" in 38, and as "bad" or "indifferent" in 33 (11 of those latter being "bad"). In these 70 cases, 8 deaths are reported, or about 1 in 9. Such figures, when compared with the rest, give no ground for asserting that multiple pneumonia is associated with exceptionally bad sanitation, while the death rate of such pneumonia is much below the average. It is remarkable that of the above 8 cases in which death occurred, the sanitary condition of the house is reported as "good" in all but two.

(c.) Upon the more difficult question as to whether the situation were exposed or confined, 753 returns show 507 to be *exposed*, or 67·3 per cent., and 246 to be *confined*, or 32·7 per cent.

The inference from these results is, therefore, that whilst there is not much difference as regards elevation and dampness or dryness of soil, there is a considerable preponderance of pneumonia in exposed places as compared with those in confined situations. This result requires to be balanced by the returns upon the meteorological conditions, to which we may now refer.

The direction of the *wind* prevailing at the time of the onset of the disease is recorded by 856 observers, as follows:—

N. wind prevailed in 38 cases, <i>i.e.</i> , in 4·4 per cent.			
N.E.	„	137	„ 16 „
E.	„	242	„ 28·3 „
S.E.	„	60	„ 7·1 „
S.	„	16	„ 1·9 „
S.W.	„	176	„ 20·5 „
W.	„	88	„ 10·3 „
N.W.	„	99	„ 11·5 „

That is to say, the E. wind was the most common, S.W. the next in order of frequency and N.E. the next; the least frequent (and probably also the least generally prevalent) direction being S. The popular belief as to the greater gravity of easterly winds (439) over westerly (303) is only borne out to the extent of the difference between 54·8 per cent. and 45·2 per cent.; whilst winds having a northerly direction (274) are nearly balanced by the southerly (252), viz., as 52 per cent. is to 48 per cent.

This question may be viewed in another way, viz., as to whether like differences prevail, and if so, to what extent, with regard to the *mortality* from pneumonia. Can it be said that one wind is more mortal in its effects than another?

N. 38 cases; 9 deaths; mortality, 23·7 per cent.			
N.E.	137	„ 25	„ 18·2 „
E.	242	„ 35	„ 14·5 „
S.E.	60	„ 13	„ 21·6 „
S.	16	„ 3	„ 18·8 „
S.W.	176	„ 32	„ 18·1 „
W.	88	„ 18	„ 20·5 „
N.W.	99	„ 29	„ 29·3 „



According to which it would appear that the N.W. enjoys the pre-eminence in this respect, whereas the E. or wind of greatest prevalence was far less deadly. The figures are, however, too small to allow of much stress being laid upon them.

Stated in more general terms, it is found that with—

Easterly winds there were 439 cases; 73 deaths; mortality, 16·6 per cent.

Westerly       "       "       363       "       73       "       "       21·8       "

Northerly     "       "       274     "       63     "       "       22·6     "

Southerly     "       "       252     "       48     "       "       19·       "

which analysis yields the somewhat unexpected result that, while the disease is more prevalent with easterly than with westerly winds, a larger fatality from pneumonia occurs during the prevalence of westerly than of easterly winds. There is not so much difference between the northerly and southerly—although the former are slightly more fatal than the latter.

Lastly, the prevailing "atmospheric condition" has been described under one or more heads by 1012 observers, with these results—

(a.) Out of 778 cases the weather is noted as being *dry* at the time of illness in 351, or about 45 per cent.; and *wet* in 427, or about 55 per cent. There are also 361 returned as "damp," but this number includes 196 also returned as "wet." Taking those returned as wet, 427, and those merely recorded as damp, 165, together, there is a total of 592 cases out of 843, or about 70 per cent., in which the condition of atmospheric humidity prevailed.

(b.) Out of 889 cases, again, the weather is returned as *cold* in 652, or 73·4 per cent., as *hot* in, 41, or 4·6 per cent., and as *mild* in 196, or 22 per cent. The term "changeable" has often been employed in association with the above, being returned in 455 cases. In 85 it is given as the sole description of the condition of weather prevailing at the time.

The general result of this part of the inquiry would seem to be that the disease is more prone to occur in exposed situations, subjected to the E. or S.W. winds, and in cold, wet (or damp) seasons.

*Abstract VI.—Family History\* of Lung Disease.*

## Pneumonia—

in the family history of 20 of series A.

„	„	„	28	„	B.
---	---	---	----	---	----

„	„	„	25	„	C.
---	---	---	----	---	----

## Phthisis—

in the family history of 51 of series A.

„	„	„	48	„	B.
---	---	---	----	---	----

„	„	„	40	„	C.
---	---	---	----	---	----

## Bronchitis—

in the family history of 19 of series A.

„	„	„	20	„	B.
---	---	---	----	---	----

„	„	„	19	„	C.
---	---	---	----	---	----

## Pleurisy—

is very infrequent, receiving mention in but six instances in all.

The above enumeration refers to—

315	cases	of	series	A.
-----	-------	----	--------	----

326	„	„	B.
-----	---	---	----

306	„	„	C.
-----	---	---	----

The chief point of interest under this heading has reference to phthisis, which is met with in a proportion of the cases varying from 1 in  $6\frac{1}{4}$  to 1 in  $7\frac{1}{2}$ , or from 17 to 13 per cent. approximately.†

Taking into consideration that the numbers have reference only to persons previously healthy, it seems probable that the tendency to pneumonia is somewhat increased by phthisical inheritance.

Of the 51 examples of pneumonia in subjects of phthisical family of series A. there died 8.

\* "Family History" includes parents, brothers, and sisters.

† Of this, as of other calculations of like kind, it is difficult to estimate the significance owing to the want of some standard of measurement. We are ignorant what is the actual proportion of phthisis to be attributed to the community at large. Dr. Reginald Thompson states that the proportion of persons giving a family history of phthisis among the policy-holders of the United States Insurance Company was 26·8 per cent. Of non-consumptives, however, less than 11 per cent. gave a family history of consumption, while of consumptives over 15 per cent. gave such a history. The numbers, however, are too few to build upon. See Dr. R. Thompson's 'Family Phthisis,' p. 17.

Of the 48 examples of the same kind of series B. there died 8.

Of the 40 examples of series C. there died 7; or 9 if two examples (old women whose precise relationship to phthisis is left uncertain) be included (Nos. 858—9).

Thus of 139 cases, 23 of direct descent from or relationship to phthisis died, or 1 in 6, a mortality not in excess of that which prevails generally.

It is quite exceptional to find subjects of pneumonia who are of phthisical family exhibiting apex pneumonia. In the few instances where this actually happens, this seat of the disease, judged by its course and termination, does not display any phthisical character.

Thus in series A. one case (No. 261) with direct phthisical descent, and two cases (Nos. 71 and 79) with indirect descent, had apex pneumonia. All recovered.

In series B. one case (No. 426), or perhaps two (No. 700), with direct phthisical descent, and five (Nos. 369, 460, 463, 575, 577 (both apices)) with indirect, had apex pneumonia. All recovered.

In series C. two (Nos. 716, 1054) with direct phthisis descent, and one (No. 806) with indirect, had apex pneumonia. All recovered.

Three cases, one for each series, having indirect phthisis inheritance exhibited an apex involved along with the base of the other lung.

From the small number of instances of pneumonia appearing in the Family History—1 in 13 upon the corrected total—it seems improbable that liability to the affection is inherited. Bronchitis is still less frequent,—less than 1 case in 15,—and pleurisy hardly appears.

#### *Abstract VII.—Previous Illnesses of Patients.*

##### Pneumonia—

in the personal history of 32, series A.

“ “ “ 34, “ B.

“ “ “ 35, “ C.

*i.e.*, in the personal history of 101 in all

##### Bronchitis—

in the personal history of 35, series A

“ “ “ 24, “ B

“ “ “ 25, “ C.

*i.e.* in the personal history of 84 in all.



## Rheumatism (chiefly acute)—

in the personal history of 16, series A.

" " " 7, " B.

" " " 11, " C.

*i.e.*, in the personal history of 34 in all.

## Scarlatina\*—

in the personal history of 9, series A.

" " " 13, " B.

" " " 11, " C.

*i.e.*, in the personal history of 33 in all.

## Measles\*—

in the personal history of 15, series A.

" " " 13, " B.

" " " 11, " C.

*i.e.*, in the personal history of 39 in all.

No other diseases appear with anything approaching the frequency of the above. Thus we have,

## Enteric Fever—

in the personal history of 3, series A.

" " " 2, " B.

" " " 6, " C.

*i.e.*, in the personal history of 11 in all.

## Tonsillitis—

in the personal history of 3, series A.

" " " 2, " B.

" " " 5, " C.

*i.e.*, in the personal history of 10 in all.

## Erysipelas—

in the personal history of 1, series A.

" " " 2, " B.

" " " 3, " C.

*i.e.*, in the personal history of 6 in all.

Pleurisy is noted in 11 cases in all.

Congestion of lungs in 9 " "

Delirium tremens in 5 " "

The above enumeration refers to—

331 cases of series A, 19 wanting.

317 " " B, 33 "

319 " " C, 36 "

Of the total corrected number, 967, suffering pneumonia, from 1 in 9 to 1 in 10 have so suffered previously.

Of the 32 of series A, who had had pneumonia before, 8 died.

\* See note to col. 10, page i., Appendix.

Of the 34 of series B, 3 died.

Of the 35 of series C, 8 died.

Thus of a total of 101 who had previously suffered pneumonia, 19 died.\*

Only 16 out of the total number of 967 notified had had pneumonia more than once before. Of these 4 died.

Rheumatism occurs rarely and very unequally in the previous histories.

The occurrence of measles and scarlatina has reference almost exclusively to children, and cannot be calculated with reference to the patients generally.

Enteric fever, tonsillitis and erysipelas are all very rare among the antecedents of pneumonia subjects.

*Abstract VIII.—Premonitory Symptoms and Onset of the Attack.*

The evidence of the existence of symptoms premonitory to those of the declared disease is difficult to weigh, for the reason that in pneumonia the actual onset is not always abrupt. Thus, if the occurrence of rigor is to be considered as marking the onset, there are many cases, about one-fifth, in which no rigor occurs. Or, if the occurrence of pain in the chest, the pleuritic "stitch" be taken, it is obvious from these returns that such a symptom often precedes the rigor, and in the view of many of our contributors the pain is regarded as prodromal. Some also have placed "fever," which precedes in some cases apparently the rigor, among the prodromata. That in many cases the disease begins insidiously, and that for days or even a week or so before symptoms declaring the true nature of the malady appear, is without question; but it is impossible for us to state precisely from the returns in how large a proportion such a mode of onset occurs. It would be undesirable to go behind these returns, and to arbitrarily decide what should or should not be considered as a premonitory symptom; and we must, therefore, be contented with an enumeration of those symptoms which have been recorded under this head. The

\* This somewhat high mortality included 2 intemperate and 2 old people (68 and 67 respectively), one of them insufficiently fed. It cannot be safely affirmed, therefore, upon the above figures, that one attack of pneumonia modifies the character of a second.

results of this portion of the inquiry ought not to be taken as strictly accurate; so much depending upon the conception formed by the individual observer as to what does and what does not constitute the "premonitory stage."

With this reservation, then, we find the following:—

	Series A.	Series B.	Series C.	Total.
Premonitory symptoms present in ...	262	231	250	743
" " absent in ...	65	94	87	246
No information in ... ..	23	25	28	76

That is to say, that among 989 cases of pneumonia, no less than 743, or 75 per cent., were marked by symptoms considered by the observers as "premonitory."

The catalogue of symptoms so recorded is a lengthy one. A large number may be considered as belonging to the doubtful class. Thus in 149 cases "pain in the chest" is given as a premonitory symptom; in 63 cases "fever;" in 74 "chilliness or shivering;" in 35 pains in the limbs, and in 16 pains in the back. No doubt in some instances these pyrexial symptoms are attributable to the premonitory catarrh, which is mentioned in 103 cases, and which includes 16 cases of bronchitis. In 29 cases dyspnœa is noted, a symptom probably directly related to the onset of the pulmonary inflammation. In 101 cases a cough was present before the declared symptoms, *i.e.*, in about one-tenth.

The remaining symptoms under this head may be roughly grouped as "general," "nervous," and "gastro-intestinal."

*General*.—Malaise, often lasting for days, led up to the attack in 107 cases. Lassitude, languor, and fatigue are mentioned in 28; and weakness or debility in 21.

*Nervous*.—Headache was present in this stage in 117 cases; more or less delirium in nine; drowsiness in six; insomnia in four; vertigo in five. In eight cases earache is noted; in two, toothache; in one, sciatica; and in one, neuralgia. Deafness in one case, and stupor in one case. In four cases, all in children, convulsions occurred; in one case apoplexy, and in another slight hemiplegia. Tremor is mentioned in one case, and "cerebral symptoms" in three. Lastly, apart from the thoracic pain and pyrexial pains, there is noted in six cases abdominal pain, and in eight pain in the joints, in two cases accompanied by swelling.

*Gastro-intestinal*.—This includes 141 cases in which vomiting



occurred, six of nausea, three of dyspepsia, 24 of purging and diarrhoea, and six of constipation.

The conditions which do not fall under either of these groups are "influenza" in one, facial erysipelas in one, albuminuria in one, sore throat and tonsillitis in 18, and epistaxis in three.

Coming now to the question of the occurrence of rigor at the onset of the disease, it is found that this symptom occurred in about four-fifths of the cases in which its presence or absence has been noted. Contrasting, as before, each series in this respect, we have—

	Series A.	Series B.	Series C.	Total.
Rigor in... ..	270	241	271	782
No rigor in ... ..	63	64	66	193
Doubtful or not noted... ..	17	45	28	90

The fatal cases amongst the 975 which make mention of this symptom include 169 deaths, *i.e.*, a percentage of 17·3, the rate being somewhat higher in the class of cases marked by rigor (142 deaths in 782), *viz.*, 18 per cent., than in that in which the rigor was absent (27 deaths in 193), *viz.*, 14 per cent.\*

Another question of some interest is whether any marked differences occur in the character of the cases of typical onset (as those commencing with rigor may be considered), and the other and smaller group. This does not appear to be the case with regard to the premonitory symptoms above mentioned, for these are fairly distributed amongst both classes, and the following figures may serve in contrasting these classes with respect to the seat of the lung inflammation:—

Rigor. No rigor.				Rigor. No rigor.			
R. lung, apex... ..	37	13		R. apex and L. base ...	0	3	
middle lobe... ..	1	1		R. mid. " " ...	—	—	
base " ...	251	61		L. apex " R. base ...	4	1	
whole ...	46	12		R. whole " L. apex ...	2	—	
L. ung, apex... ..	21	7		" " L. base ...	23	4	
base ...	187	47		L. mid. " R. " ...	1	—	
whole ...	34	7		L. whole " R. apex ...	4	2	
Both lungs, apices ...	1	2		" " R. base ...	21	1	
bases ...	139	29		Whole of both lungs ...	8	2	
Disseminated areas of				"Central" ...	—	1	
both... ..	1	—		No part ...	1	—	

\* It is, of course, likely that among the 90 returns which are defective on this point, and which include 23 fatal cases, several may belong to the series of "no rigor," in which case the above reckoning may not represent the actual truth.

*Abstract IX.—Part of Lung affected.*

	Both sides.										Right side.				Left side.													
	Both bases.		Right base, left apex.		Left base, right apex.		Right whole, left base.		Left whole, right base.		Both ap'ces.		Both wholes.		Right whole, left apex.		Left whole, right apex.		Totals both sides.	Base.	Apex.	Whole.	Totals right side.	Base.	Apex.	Whole.	Totals left side.	Wanting or ambiguous.
Series A.	66	2	1	9	11	1	3	1	4	98	106	17	18	141	89	11	12	112									?	
Series B.	56	0	2	13	5	2	1	1	1	81	115	21	24	160	79	7	14	100									"Right base left middle" 1. "Left base right middle" 1. "No part" 1	
Series C.	57	5	1	6	9	1	7	0	1	87	110	21	23	154	83	12	20	115									Left side then right 1. Patches of both 1. Wanting or ambiguous 4.	
Grand Total.	179	7	4	28	25	4	11	2	6	266	331	59	65	455	251	30	46	327										

*There died of the above:—*

Series A.*	22	0	0	2	2	1	3	0	2	32	15	1	5	21	11	2	3	16									
Series B.†	10	0	0	5		0	1	1	1	21	14	3	5	22	8	2	3	13									
Series C.‡	19	1	0	3	1	0	4	0	0	28	12	3	8	23	10	1	2	13									"Right middle" 1.
	51	1	0	10	7	1	7	1	3	81	41	7	18	66	29	5	8	45									

The right lung is more often affected than the left, the left more often than the two together.

\* Of these 8 were young children or old people; 4 were intemperate and one insufficiently fed.

† Of these 3 were young children and a man of 74: 3 were intemperate.

‡ Of these 3 were old men and a child under 4: 3 were intemperate.

The most common seat of pneumonia is the right base,\* which is the part affected in between one-third and one-fourth of the whole number. The least common seats are the two apices together, and the two lungs together, otherwise than as to their bases, *e.g.*, either the base or the whole of one lung with the apex of the other; (the whole of one lung with *base* of the other is somewhat more common, about equal in frequency to the left apex).

Next in order of frequency to the right base is the left base, and next again both bases. Then, with considerable interval come the whole of the right lung, the right apex, the whole of the left lung, the left apex.

The proportion of deaths for the several parts of the lung respectively can only be estimated very generally, and with reference to the higher numbers. The rate of mortality where both lungs are involved is the highest of all, and more than double that where one side only is affected, the proportion being 7 to 3·3. There is no appreciable difference in respect of mortality between the two sides, 1 to 7 representing the death rate for each respectively. Mortality of right base is 1 in 8, of left nearly 1 in 9. The highest rate of mortality with reference to site next to that attributable to both sides belongs to both bases, the next highest to the whole of the right lung. Yet here, as well as in the case of the apices, the numbers become too small for trustworthy conclusions and very dissimilar for the two sides. All that can be said is that affection of the whole of one lung is fatal in 1 in 4·3 instances (upon the showing of 111 cases), and affection of one apex fatal in 1 in 7·3 instances (upon the showing of 89 cases†).

\* The base of the lung must be taken to indicate its lower part merely. The returns do not enable us to determine the precise locality which is first attacked. It is observable that the whole of both lungs (by which we are to understand the greater part of both) receives mention much more often than does one lung together with the apex of the other.

† The 28 apex pneumonia of Series A., includes as many as 9 children. Three of the 28 died. A like number of apex pneumonia of Series B., includes 7 young children and 2 aged 12. Five of the 28 died. The third series has 33 with but 3 young children and 1 child of 12. This last series, therefore best represents apex pneumonia as commonly understood. The deaths are but 4 (1 in 8). Thus neither in its mortality, nor, as will be shown presently (p. 50), in its duration does apex pneumonia compare unfavourably with base pneumonia.



*Abstract X.—Duration of Fever.*

Days:—	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17, 18	over
Series A .....	3	12	19	22	34	60	31	18	28	7	11	2	9	1	4	7	6
Series B .....	1	4	12	27	26	55	39	15	35	11	14	3	16	2	3	2	13
Series C .....	0	9	18	45	25	36	37	27	21	10	14	6	9	2	0	3	15
	4	25	49	94	85	151	107	60	84	28	39	11	34	5	7	12	34

*Days of Crisis.*

Days:—	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17, 18	?	over.
Series A .....	2	5	12	14	22	32	16	8	5	4	0	1	1	0	0	1	1	(21d)1
Series B .....	1	3	10	17	13	32	21	8	10	1	2	1	0	0	0	0	2	2
Series C .....	0	7	13	28	17	21	15	13	6	1	1	2	0	0	0	0	2	4
	3	15	35	59	52	85	52	29	21	6	3	4	1	0	0	1	5	

Taking duration of fever without reference to its mode of termination, whether sudden or gradual, the 7th is by far the most common day of subsidence. This, true in the aggregate, is true also of series A. and B. Series C., however, shows the 5th as the favourite day of termination, and is further exceptional in giving to the 8th day the same frequency as the 7th.

All that can be said, therefore, as applicable to the three series is as follows:—

The subsidence of fever in pneumonia, whether sudden or gradual, is apt to fall between the 4th and the 10th day, the 5th, 6th, 7th, and 8th days having predominance. On the 11th day there is a marked fall. Subsidence on the 12th day, although quite exceptional, is less uncommon than on the 11th. Subsidence on the 13th day is extremely rare. It is much commoner on the 14th, which has nearly equality in this respect with the 12th day.

Subsidence at any later period than the 14th day is very unusual, and as regards *sudden* subsidence, so rare that it need not be considered.

If series C. be excluded, subsidence of fever on the 7th day would exceed in frequency the like event for both 5th and 6th

days, taken together. The 8th day, although rivalling the 5th and 6th, taken severally, falls off considerably from the 7th. The 10th day outnumbers the 9th and the 11th put together as the date of fever subsidence.

Thus the 9th, 11th, 13th, and 15th are all remarkable for infrequency of fever subsidence, as compared with the days on either side of them.

This last observation indeed applies to series C. likewise, except as regards the 9th day.

Speaking generally, it would appear that taking the odd numbers from 5 to 15 inclusive, the first two, 5 and 7, indicate favourite days of subsidence, while the last four, 9, 11, 13, 15, indicate rare days of subsidence.

Sudden subsidence of fever applies, as has been seen, to a minority of the whole number of cases. This mode of termination, although not necessarily "critical" in all instances, may perhaps be safely taken to indicate generally the days of crisis. Understood in this sense, the 5th, 6th, 7th, and 8th are the favourite days of crisis, 5th and 6th contending doubtfully together for a majority. The 7th day is far ahead of the rest in the aggregate as well as in each individual series, with the exception of series C., where it is slightly outnumbered by the 5th day. After the 8th day (which on the whole has an equality with the 6th as a day of crisis), there is a considerable falling off in the numbers. And the 10th day, which shows prominently as the one for fever *subsidence*, is but rarely a day of crisis. From the 11th to the 14th day subsidence by crisis is extremely rare.\*

\* The fact that the large majority of cases of pneumonia here summarized terminate within ten days would seem to dispose of one objection to this kind of investigation, viz., that errors of diagnosis must be frequent, and especially that pneumonia is likely to be confounded with enteric fever. The absence of sequelæ is a further proof of the like kind.

It may be of interest to compare our figures in respect of critical days with those of other countries. Those of Bleuler, Wunderlich and Ziemssen are perhaps the best known. Their observations refer respectively to 146, 75, and 107 cases. In all three *the seventh* (as with us) is the most frequent day, numbering about a quarter of Bleuler's and a third each of Wunderlich's and Ziemssen's. But with this all that is common to the three is expressed. The 5th is indeed a frequent day with all. But while with Wunderlich the 6th day equals the 5th in frequency and with Bleuler slightly exceeds it, Ziemssen finds the 6th

*Abstract XI.—Termination of Fever.*

## SUDDEN—GRADUAL—FATAL.

There terminated *suddenly*—

Of series A	...	...	...	...	125
„ B	...	...	...	...	124
„ C	...	...	...	...	126

There terminated *gradually*—

Of series A	...	...	...	...	153
„ B	...	...	...	...	164
„ C	...	...	...	...	168

There *died*—

Of series A	...	...	...	...	69
„ B	...	...	...	...	58
„ C	...	...	...	...	65

In this enumeration 2 are wanting in series A.

4	„	„	B.
7	„	„	C.

Taking the whole number of cases together, gradual subsidence is more common than sudden subsidence, in a proportion of about 4 to 3, a proportion which may be said to apply pretty nearly to each of the three series. Nevertheless, the proportion a most infrequent day of crisis, only 5 of his 107 falling on that day. Again the 8th, which is a day of crisis in a sixth of Bleuler's cases (exceeding the 5th in frequency), occurs but four times in Ziemssen's 107 and but four in Wunderlich's 75.

Speaking generally, Bleuler makes rise by strides up to the 7th day and then subsides gradually to the 9th when, except for the 10th and 12th days, he disappears altogether. Wunderlich, rising earlier, but more gradually, up to the 7th day, then falls abruptly and finally disappears on the 9th day. Ziemssen has the uneven days conspicuous, especially the 5th and 7th, and (for their position) the 9th and 11th. In the infrequency of his 4th and 6th days he is in direct opposition to the other two. The three series are as follow :—

Bleuler.		Wunderlich.	Ziemssen.
3rd day	6	10	9
4 „	13	11	3
5 „	22	14	31
6 „	26	14	5
7 „	32	19	35
8 „	24	4	4
9 „	12	3	9
10 „	6	0	0
11 „	1	0	8
12 „	3	0	0
13 „	1	0	3
	146	75	107



of gradual to sudden subsidence varies very widely in consecutive groups of cases. Gradual subsidence comprehends almost all the cases whose duration exceeds ten days. This mode of termination characterizes pneumonia of both bases more particularly, where it applies to nearly three-fourths of the recovering cases. Pneumonia of the right base has gradual subsidence in about half the examples of that kind. The same may be said for the left base.

Sudden subsidence (as well as short duration) is seen especially in apex pneumonia. Thus, in the 33 cases of that sort of series C., there are no less than 18 with sudden to half that number with gradual subsidence. Similarly in series B. instances of sudden subsidence are greatly in excess of the gradual.

In pneumonia of both bases we have not only gradual subsidence of fever greatly in excess of its sudden subsidence; we have, moreover, a longer average duration and a much higher mortality than elsewhere. Thus, of the 66 examples of both bases pneumonia (series A.), 29 had gradual, 15 sudden subsidence, 22 died. Of the 54 similar examples (series B.), 30 had gradual, and 14 sudden subsidence, 10 died. Of the 56 (series C.), 22 had gradual, 14 sudden subsidence, and one doubtful; 19 died.

These, along with other characters of "double pneumonia," lead us to suspect that a proportion at least of the cases so described in the Report are not proper illustrations of pneumonia in the precise sense contemplated by the present inquiry. At all events, the rejection of cases of this class (which are very unequal in the three series) tends to uniformity, as will be seen below.

*Termination of Fever—Sudden, Gradual, and Fatal,  
excluding instances of "Both Bases."*

Series A., sudden	...	...	...	125 - 15 = 110
gradual	...	...	...	153 - 25 = 128
fatal	...	...	...	69 - 22 = 47
Series B., sudden	...	...	...	124 - 14 = 110
gradual	...	...	...	164 - 30 = 134
fatal	...	...	...	58 - 10 = 48
Series C., sudden	...	...	...	126 - 14 = 112
gradual	...	...	...	168 - 22 = 146
fatal	...	...	...	65 - 19 = 46

A glance at the Returns will show the very unequal incidence of death in successive pages, varying from 1 in 3 (p. lxxxviii.-ix.) to 1 in 40 (pp. xlv.-vii.). Yet when large numbers are taken these inequalities disappear, and the gross death-rate does not differ widely for the three series.\* Even if these be subdivided the mortality of the two halves of series B. and C. respectively, are almost precisely equal, while the inequality of the two halves of series A. (which is considerable, 28 to 41), is accounted for by the exceptionally low mortality already alluded to of 40 cases between Nos. 412 and 451.

\* The rate of mortality of the several series of the Report varies from 1 in 6 to 1 in 5, or from 16 to 20 per cent.

We may compare this with the mortality at some of the London hospitals for the year 1883 :—

	Per cent.
Guy's, 62 cases with 10 deaths .....	16
St. Bartholomew's, 137 cases with 28 deaths .....	20·4
London Hospital, 100 cases with 20 deaths.....	20
St. George's Hospital, 86 cases with 18 deaths .....	20·9
Middlesex Hospital, 66 cases with 7 deaths .....	10·6
Westminster Hospital, 38 cases with 4 deaths .....	—

Such other returns as we have received either refer to a less recent period or the precise dates are wanting. From the statistics of consecutive years, however, we are able to say that the death rate of pneumonia varies very widely from year to year. Thus, in 1875 the mortality of pneumonia at the London Hospital was not quite 24 per cent., in the year following it was nearly 39 per cent. Again, of 40 cases admitted into Middlesex Hospital in 1871, 14 died, while of the same number admitted in 1873 only 5 died. Similarly, the death-rate at St. Bartholomew's varies from 16 per cent. to 20 per cent. At the Westminster Hospital it varies from 10·6 per cent. to nearly 23 per cent. The lowest mortality obtained from any one general hospital is 10 per cent; the highest 39 per cent; but these are both exceptional.

In the case of children, taking croupous as distinguished from catarrhal pneumonia, the death-rate is exceedingly small. At the General Hospital for Sick Children, Pendlebury, only 3 deaths occurred out of 144 cases admitted in 3 years (1881—3).

The numbers dealt with in our Report are far larger than any to be met with elsewhere, and we believe that its aggregate mortality fairly represents the present death-rate of acute pneumonia.

It is not without interest to compare these figures with the old statistics of Chomel, Louis and Andral, showing a mortality varying from 30 to 55 per cent.; or again with Bouilliard's "jugulant" practice with an asserted death-rate of about 12 per cent., and the late Dr. Hughes Bennett's 129 cases with only 4 deaths.

We have here to acknowledge the courtesy of the Medical Registrars of the several metropolitan hospitals, and of Dr. Ashby, of Pendlebury Hospital, in supplying us with the above particulars.

The days on which death happened were as follows:—

			Series A.	Series B.	Series C.	Totals.
There died on the	1st day	...	—	—	1	1
"	2nd "	...	—	—	—	—
"	3rd "	...	1	1	3	5
"	4th "	...	4	4	3	11
"	5th "	...	5	0	8	13
"	6th "	...	6	6	7	19
"	7th "	...	12	7	7	26
"	8th "	...	10	9	6	25
"	9th "	...	3	2	4	9
"	10th "	...	6	8	5	19
"	11th "	...	2	5	4	11
"	12th "	...	0	2	2	4
"	13th "	...	3	3	1	7
"	14th "	...	2	—	—	2
"	15th "	...	1	—	1	2
"	16th "	...	—	1	2	3
"	17th "	...	1	1	1	3
"	18th "	...	—	—	—	0

Later than this deaths for each series occur as follows:—

19th, 22nd, 38th, and 62nd ... series A. 1 for each.

19th, 20th, 24th, 26th, 35th, 37th, and 46th „ B. „

21st, 28th, 32nd, 70th ... „ C. „

The majority of deaths from pneumonia fall from the 4th to the 11th, viz., 133 out of the 175 where we have information upon this point. The 6th, 7th, 8th and 10th are the days on which death is most common. The 5th day earns its place by virtue of the comparatively large number of such fatal cases in series C. There is not one for the previous series. After the second week death becomes very infrequent. From the 14th to the 21st there are but 14 deaths in all for the three series. Deaths after lengthened periods of illness, four weeks and over, number eight. They are made up as follows:—

Series A. 38th day (54), 62nd day R. b. intemp. (307), phth. L. b.

Series B. 37th day (363), 35th day R. l. wh. (459), R. b. phth. ?

46th day (661), gangrene, R. b. intemp.

Series C. 32nd day (758), 70th day R. l. (796), phth. R. b.

28th day (992) Both b.

Thus two, if not three of the eight, were examples of phthisis; one was a case of gangrene in an intemperate man.



*Abstract XII.—Sequelæ.*

## Chronic pneumonia—

was sequela in 7 of series A.  
 „ „ 7\* „ B.  
 „ „ 5 „ C.  
*i.e.*, in 19 or 20 of the whole.

## Empyæma—

was sequela in 2 of series A.  
 „ „ 2 „ B.  
 „ „ 1 „ C.  
*i.e.*, in 5 of the whole.

## Pleurisy—

was sequela in 2 of series A.  
 „ „ 5 „ B.  
 „ „ 1 „ C.  
*i.e.*, in 8 of the whole.

## Debility—

was sequela in 1 of series A.  
 „ „ 11 „ B.  
 „ „ 2 „ C.  
*i.e.*, in 14 of the whole.

## Cough—

was sequela in 3 of series A.  
 „ „ 7 „ B.  
 „ „ 2 „ C.  
*i.e.*, in 12 of the whole.

## Bronchitis—

was sequela in 3 of series A.  
 „ „ 0 „ B.  
 „ „ 7 „ C.  
*i.e.*, in 10 of the whole.

## Taking the three series together—

Pericarditis was sequela twice.

Endocarditis „ once.

Tonsillitis „ twice.

Otorrhœa „ twice.

Acute mania „ twice.

Delirium tremens „ twice or thrice.

\* Or 8 if “dulness and cough,” one case, be included.

Other sequelæ are herpes zoster (2), œdema, from thrombosis (1), meningitis (1), jaundice (2), suppuration of external ear (1), slight deafness (1), congestion of brain (1), gastric disturbance (1), abscess in axilla (1), myalgia (1), abscess in axilla (1), abscess in leg (1), loss of hair (1), sweating (1).

In a large number of instances pretty evenly distributed throughout the series (about 66 in the first, 59 in the second, and 62 in the third), no information is given under this heading. Yet inasmuch as the actual duration of these cases, with the mode of termination, whether in recovery or death, is duly stated, it may be fairly assumed that sequelæ were absent in those examples, although the negative fact is left unrecorded.

If that be so sequelæ are of very rare occurrence in pneumonia. Phthisical destruction occurred but in four cases, one, if not two of these, having phthisis to start with, and two men, one a drunkard, died of gangrene of the lung.

In 19 or 20 cases the affected lung had not wholly recovered at the time of last observation; and 14, all but three of these being in series B., were slow in recovering strength. Pleurisy and empyæma together, are sequelæ in but 13 instances.

The occurrence of acute mania among the sequelæ of pneumonia (rare as it is like all the rest), deserves notice.

### *Abstract XIII.—Treatment.*

Some information as to treatment has been furnished in 1037 returns, with more or less precision. Amongst these are a certain number in which important omissions have been made, *e.g.*, as to whether or not stimulants were prescribed, or what was the line, if any, of medicinal treatment. This fact deprives the numerical analysis of some of its importance, but nevertheless as an indication of the modes most generally adopted it is probable that this section of the Report, which, for want of space finds no place in the Table, may not be devoid of interest.

Commencing first with local measures, 287 mention the application of poultices to the chest, and 117 the employment of counter-irritants. In 18 it is stated that leeches were applied;

in one instance to the temple for the relief of headache, but in most cases more for the relief of the "stitch" in the side than for any ulterior reason. In one case "local bleeding," and in two cases wet-cupping were employed. (For application of cold *v.* Antipyretics).

Inhalations were practised in 15 cases.

As to diet, there is a consensus of opinion in favour of this being good, nutritious, and ample.

A certain number draw attention to the importance of maintaining a constant temperature in the sick room, with good ventilation, and some adopt measures for ensuring a warm moist atmosphere around the patient.

The treatment is said to have been "expectant" in 134 returns; and under the same head it is perhaps justifiable to include the administration of "salines," which are mentioned in 127 cases. It should be remarked that under "expectancy" doubtless many of the other lines of treatment mentioned below are to be included, and probably were so included by those who employ this term as conveying best the idea that the measures taken were rather symptomatic than otherwise. And, indeed, if this view be correct, by far the majority of practitioners carry out the "expectant" plan in treating this disease.

The terms "restorative" and "supporting" are used by 16 observers, and that of "stimulant" by 78. But very many others indicate the nature of the means employed in the last-mentioned direction. Thus, the diffusible stimulants, as carbonate of ammonia and ether (mainly the former), were prescribed in 250 cases, the ammonia being also of value as an expectorant, and frequently prescribed in combination with other expectorants. Indeed, this is the drug which is far more commonly employed than any others. Alcohol, in one or other forms, mostly brandy (then whiskey, port wine, champagne), is mentioned in 424 cases; whilst in 104, or one-tenth of the whole number, it is expressly stated that "no stimulants were given."

In 94 cases antimonials were prescribed, sometimes pushed to produce depression, often apparently more as an addition to other diaphoretics, which are recorded in 144 instances. Diuretics are only expressly mentioned in 12 cases. Expectorants, such as ipecacuanha, ammonia, squills, senega, &c., are stated in



214 instances, mostly reserved for the later stages of the attack, salines and diaphoretics being prescribed at first.

Sedatives, as opium in 60 cases, bromides in 14, and occasionally chloral, are employed to procure sleep and abate delirium. It is unlikely that these figures at all represent the frequency with which they are given.

In 42 cases mention is made of iodide of potassium, prescribed in the majority, but not in all, late in the disease, to "aid in resolution." Tonics, as iron, bark, mineral acids, &c., are mentioned in 78, as being given during convalescence.

The term "antiphlogistic" is employed by 22 observers, mostly without explanation. Calomel is mentioned in about ten returns, and "venesection" in only two (in Nos. 165 and 864).

The modern phrase, "antipyretic," has replaced that just quoted, and is given as solely descriptive of the treatment employed in seven instances; but "antipyretic" measures, as judged from the prescriptions, are very largely in favour. Thus quinine is to be found in 143 returns; aconite in 125; digitalis in 65; salicylic acid or its compounds in 41; veratria in 4; cinchonidine (5 grains during eight hours) in one (No. 737), and tartrate of chinoline in one, "given until the temperature became normal" (No. 115).\*

Cold sponging was resorted to in nine cases. In one (No. 716) where salicylate of soda was also given, it is said that "cold sponging was tried one day, but as result was not satisfactory it was discontinued." In another (No. 759) the reporter says:—"Sponging with cold water three times during twenty-four hours effected a lowering of temperature and was generally beneficial. Quinine, gr. i., three times daily, when sponging stopped on fourth day." Cold compresses or applications to the chest are only mentioned in four cases; to the head, in six. In five cases the applications were warm or tepid; in three warm or tepid baths were given, and in two vapour baths.

\* Aconitine, veratrine, arseniate of strychnia, digitaline, were prescribed in three cases by one observer. In one other return occurs the statement that "during pyrexia aconitine, veratrine, and digitaline were given; these soon reduced the fever." The lately introduced substance, "kairin," a derivative of chinoline, is not prescribed in any case.

Lastly, to mention some drugs not included in the foregoing summary—ergot was given in nine cases; belladonna in three; phosphorus in two; phosphate of ammonia in two; hydrastin in one (No. 963); pilocarpin in one; and colchicum in one.

It is manifest that the lines of treatment are so various as not to permit of any value being attached to a comparison of mortality.

*Abstract XIV.—Analysis of Foot Notes.*

*A. Anomalous Symptoms and Sequelæ.*

In series A, 2 (66, 134) had relapse. Both recovered.

1 (38) a second and a third attack with fatal termination.

1 (58) had profuse epistaxis the day before crisis.

1 (182) had "brain symptoms at the outset which were relieved on appearance of physical signs 48 hours later."

2 (32, 149) had acute mania following. In the first (a temperate man of 22) on the 11th day; in the second (a man of 19, a total abstainer) along with the pneumonia.

1 (301) had maniacal delirium with slow pulse and low temperature 2 days after crisis.

In series B, 2 (654, 672) suffered relapse. Both recovered.

1 (508, a child of 7) had epistaxis on the 5th day with fall of temperature; in 1 (629) delirium tremens supervened; 1 (503) ended in phthisis.

Of the fatal cases having unusual symptoms 2 (501, aged 54; 661, aged 60) died of gangrene of the lung; both men, the latter intemperate. A fatal pneumonia of right apex occurred in an old man of 70 (385) during an attack of acute mania. A temperate man of 42, convalescent from pneumonia (488); suddenly fell dead while dressing.

In series C, 1 (706) suffered relapse.

1 (730) had delirium tremens concurrently and died; and 1 (796) "ran on to phthisis, of which he died."

B. *Pneumonia ascribed to other Causes than Chill or Exposure.*

	Infectious origin.	Pytho- genic origin.	Nervous origin.	Noxious inhalation or expo- sure.	Bodily injury.	Alcohol.	
						a. with exposure.	b. without. exposure.
Series A.	8		—	—	97		
	12	12	—	—	178	a. 44	b. 63
	28	—	—	—	—	150	—
	52	—	—	—	—	—	165
	98	—	—	—	—	—	—
	100	113	—	—	—	—	—
	125	140	173	—	—	—	—
	160	225	271	—	—	—	—
	169	226	—	—	—	—	—
	204	290	—	—	—	—	—
	240	—	—	—	—	—	—
	301	—	—	—	—	—	—
	330	—	334	—	—	—	—
	336	336	336	—	—	—	—
	—	—	342	—	—	—	—
Series B.	408	400—3	495	417	453	—	—
	475	475	534	421	—	—	540
	479	479	549	566	—	—	—
	523	—	559	627	—	—	—
	583	—	685	—	—	—	—
	587	587	686	—	—	—	—
	—	—	689	—	—	—	—
	—	—	—	—	—	—	—
Series C.	720	—	—	711	—	—	723
	832	—	—	—	—	756	730
	858	858	804	917	—	—	762
	859	859	995	919	—	—	878
	864	—	—	976	—	—	905
	866	866	—	—	—	—	971
	992	931	—	—	—	—	1038
	1011	992	—	—	—	—	—
	1017	1025	—	—	—	—	—
	1018	1026	—	—	—	—	—
	—	1027	—	—	—	—	—
	—	1028	—	—	—	—	—
	—	1029	—	—	—	—	—
	—	1046	—	1043	—	—	—
	—	1063	—	1044	—	—	—
	—	1064	—	—	—	—	—

\* \* The numbers in leaded type refer to cases which appear under more than one column, the category to which they properly belong being in doubt. See further on this subject, 'Note on the Etiology of Pneumonia,' p. 60.

In series A there are 14 cases of pneumonia of supposed *infectious* origin. Of these there died 3 (12, 204, 240); 2 of these (*vide* Table above) are ambiguous, of whom 1 (12) died.



In series B are 6 such cases. Of these there died 2 (408, 583). 3 of these are ambiguous; none of whom died.

In series C are 10 such cases. Of these there died 3. 3 of these are ambiguous of whom there died 2 (858, 859).

Thus, if all the cases be included, we have 30 examples of infectious pneumonia with 8 fatal. But if ambiguous cases be excluded, we have 20 examples of infectious pneumonia with 5 fatal.

In series A there are 7 examples of pneumonia of supposed *pythogenic* origin. Of these there died none. 2 of the 7 are ambiguous (*vide* Table).

In series B are 7 cases. Of these there died none. 3 of the 7 are ambiguous.

In series C are 13 cases. Of these there died 3 (858, 859, 992). 3 of the 13 are ambiguous, of whom there died 3.

Thus, if all the cases be included, we have 27 examples of *pythogenic* pneumonia with 3 fatal. But, if ambiguous cases be excluded, we have 19 examples of *pythogenic* pneumonia, none of whom died.

The numbers are insufficient for any reliable conclusion, but they are in harmony with other evidence of the Report, that pneumonia of this origin is of low mortality.

Of pneumonia of *nervous* origin (that is pneumonia associated with fatigue, mental distress or anxiety) there are in all without separating the series, 14 cases where that is alleged by the observer as cause or part cause of the illness (one of them, non-fatal, being ambiguous). As many as 9 died, *viz.*, 271, 342, 495, 534, 549, 559, 686, 804, 995, *i.e.*, all but 4, or 5 if we include the one of doubtful origin.\*

Of pneumonia associated with injury there are but 3 examples. 1 (97) was "caused by injury to chest wall. 1 (178) arose 11 days after amputation of the breast; and 1 (454) "while the patient was in bed with broken leg." All recovered.

Of pneumonia originating in *alcoholic excess* there are 13 well marked examples. In 3 of these exposure, arising out

\* This high mortality appears the more remarkable when the fatal are cases analyzed. All are temperate and sufficiently fed. Two are total abstainers. None are advanced in life (unless one aged 62 be so accounted). Bodily fatigue is alleged in as many as 4. Anxiety or mental depression in 4, grief in 1.

of the drunkenness, is alleged as the exciting cause. One of these 3 died. In 10 there is no mention of exposure, but only "heavy drinking," "drinking bout," "lived chiefly on alcohol," "drinking for a month," &c., &c. (See Nos. 63, 723, 730, 762, 878, 905, 971, 1038.) They are all young or middle aged, the oldest 50, the next oldest 41, the youngest 22. *All are men.* All but 2 (165, 540) died.

The column headed "noxious exposure," is made up as follows:—

5 (417, 566, 711, 1043, 1044) were exposed to the hot sun.

1 (421) exposed to dust in his employment.

1 (917) "half stifled with smoke."

1 (919) living in a flooded country in house "where all other members of the family had ulcerated throats."

All of these 8 recovered.

1 (627) immersed in dock water\* and another (976) "exposed to furnace heat" represent the fatal cases under this heading.

#### *Note on the Etiology of Pneumonia.*

IN March, 1884, a further note was published in the *Association Journal* directing attention to the central point of this inquiry—viz.,—the etiological problem, with a view of eliciting further information. Several statements were received in response to this invitation; the more detailed and important of them may here be briefly summarised; but many have to be omitted for want of space.

1. *T. J. Ollerhead, Minehead, Somerset.*—From ten years' experience, states that pneumonia is not prevalent in the district. Most cases occur in the early spring, attributable, 1st, to the east wind then prevailing, and, 2nd, the depression of vitality, due to the winter months, and therefore greater susceptibility to such influence. Two instances are given—one of a sailor's wife who fell ill after exposure to the east wind by opening the door at night—a fatal case; the other, that of a keeper who, after the fatigue of a day's sport, returned home in the cold night. The writer believes that the factors at work are—a constitutional debility or a local lung weakness, and then exposure to the east wind. He notes that the greatest mortality occurs in houses having an east aspect.
2. *T. Corbett, Kingston-on-Thames, Surrey.*—Gives instances of apparent contagion and pythogenic origin. A. Three cases occurred in one house in January 1884; B. Two cases, marked by typhoid symptoms, in one house in March.

\* Note appended to this case by Dr. Allan may here be re-quoted. "It has been observed in Liverpool that almost all cases of immersion in the docks die of pneumonia, while those who fall into the river usually recover; the foulness of the dock water is supposed to explain this.

Drains had been recently opened in the vicinity. In four of these five cases the onset was preceded by severe catarrh. Believes that pneumonia is always preceded by a blood-change (probably through agency of nervous system), induced by chills, fatigue, mental worry, intemperance, alcohol, gout, &c. Has observed that in subjects of Bright's disease the pneumonia is of the "spreading" character; and that intemperance disposes to severe forms of the disease. Has no decided views upon atmospheric influences.

3. *J. T. George, Keith, Banffshire*.—Apparent contagion—(No. 747 in Tablo). A farmer "took cold" at market, ten miles from home; on arrival, had pain in side, fever, thirst. On the fifth day of his illness his wife and one of the family fell ill; two days later a servant; and then other members of the family were attacked. All were typical cases of pneumonia. The locality is healthy. In another case a servant girl was attacked on Dec. 4, 1882, with pneumonia; six days later her mistress fell ill. The latter's husband was attacked with same disease four days after his wife, at the house of a friend whither he had removed. Eight days later his friend's daughter, and later the parents also, were attacked.
4. *T. H. Redwood, Rhymney, Mons.*—Notes that the worst cases occur amongst the Irish, and are explained by insanitary dwellings, drink and exposure. He has observed that pneumonia often begins with congestion of the liver; and after a drinking bout. That it is sometimes epidemic; a character which is determined, as he believes, by atmospheric conditions.
5. *R. Ross, Lochs, Stornoway, N.B.*—Considers the principal causes of pneumonia in persons previously in good health to be—I. *Predisposing*. 1. Constitutional peculiarities, and family tendencies. 2. Debility and functional derangements from fatigue, anxiety, want of sleep, alcoholic excess, or—3. Previous attacks. II. *Exciting*. 1. Certain atmospheric conditions observable in seasons when pneumonia, erysipelas, puerperal inflammation, &c., are prevalent and apt to be epidemic. 2. Exposure to cold (quite insufficient in ordinary circumstances to produce any serious effects) in predisposed subjects. Gives instances of pneumonia following chilling of body when overheated. Has not seen any case of presumed infection.
6. *W. R. Sergeant, Crowland, Peterborough*.—In majority of his cases tolerably clear evidence of exposure to cold. In the spring of 1883 had many cases of acute pneumonia "consequent upon the cold winds, chiefly north, then prevailing,—eleven cases in five weeks, many fatal." Also refers to two instances of apparent contagion. 1. A woman, E. F., fell ill on May 13th; the woman who nursed her was attacked on the 26th and died on the 29th from double lobar pneumonia. 2. "M. P. came here from a neighbouring town to see her brother dying from pneumonia; a day or two after his death and while still here she developed pneumonia."
7. *C. H. W. Parkinson, Wimborne, Dorset*.—Isolated or ordinary cases with history of exposure of milder type than "epidemic" cases. In latter recovery more protracted; and there is tendency to extend to both lungs and to relapse. "Often two cases in one house, and several more in immediate locality. Prevailing wind easterly with hot sun and cold nights, following on southerly winds with damp weather." Sometimes apparently communicable; case of mother dying from pneumonia shortly after nursing her son during an attack. About the same time four other cases in vicinity. Of these six cases—three dwelt on hill and three in valley on marshy soil. Insufficient food, intemperate habits and bad sanitary arrangements aggravate but do not cause the disease.
8. *T. F. Pearce, Haslemere, Surrey*.—Cases more frequent in spring of year. Not seen any epidemic prevalence, nor had any evidence of infection. As to pythogenic origin, has more than once met with pneumonia where the subject had been exposed to an atmosphere of animal putrefaction; and in this class the general symptoms are out of all proportion to the local.



9. *T. F. Raven, Broadstairs, Kent.*—Thus summarises etiology of 27 cases he has reported (v. Table):—

*Definite Causes.*

1. Drink ... ..	4
2. Insanitary state of house ... ..	5
3. Drink and deficient food ... ..	1
4. Drink and destitution ... ..	1
5. Communicated ... ..	1
6. Exposure ... ..	1

*Suspected Causes.*

7. Unhealthy neighbourhood ... ..	3
8. Occupation of patient ... ..	1
9. Insanitary house ... ..	1
Causes altogether doubtful ... ..	9

10. *J. A. Erskine Stuart, Balley.*—A number of cases in this district among the "blanket-raisers;" much brimstone used in storing the blankets. The clay subsoil of the district may predispose.
11. *A. Sutherland, Invergordon, N.B.*—Prevalence of pneumonia influenced by N.E. winds. Most common assignable cause is exposure to cold and wet. Contagion possible in some cases. During last year (a year more abounding in pneumonia cases than the whole ten previous years put together) had 4 patients with pneumonia in one house (mother first, then 2 children, then father, within five weeks), 3 cases in another, 2 cases in three different houses.
12. *J. Neil, Portsmouth Borough Asylum.*—Alludes to prevalence in the insane; mostly in chronic dementia with sluggish peripheral circulation. Has seen cases among subjects of acute mania, in some instances due to exposure. Not met with cases of infection.
13. *W. S. W. Vaughan, Crewe, Staffordshire.*—Has often had cases which seemed to be caused by insanitary conditions, similar to those producing enteric fever and some forms of diarrhoea.
14. *W. E. Green, Sandown, Isle of Wight.*—Pneumonia, as a rule, is the result of chill; occurring most during prevalence of cold east wind, or during north-west wind, which is often colder here than the east. During past six months no case met with in his practice, an immunity attributed to mild winter.
15. *A. C. Graham, Weybridge, Surrey.*—In case reported (537), two or three dwellers in same house ill at same time with catarrhal attack.
16. *J. Edwards, Liverpool.*—In one of his cases reported (968), a servant girl, whilst menstruating "took cold" by washing steps of house on cold, wet, and windy day. Another patient was working in the sewers, and was taken suddenly ill with great prostration, jaundice, vomiting, purging, and hæmaturia. The jaundice disappeared in a few days, and he then went through an attack of pneumonia.

17. *A. de W. Baker, Dawlish*.—In one case reported (167), the cause assigned was exposure to cold. In another (971) intemperance, or rather sudden withdrawal of habitual supply of drink. Mentions fact of two cases of acute pneumonia in same lane, another nursing her son was attacked.
18. *G. H. Darwin, Didsbury, Manchester*.—Has met with pneumonia following (*a*), catarrh, (*b*) infection; *c. g.*, 2 or 3 additional cases, if not in same house in same locality, (*c*) traumatism. In February, 1882, a lad received a blow on chest, followed by pneumonia (*Lancet*, Sept. 23, 1882); and a case has also been seen of a lady, after carriage accident.
19. *J. M. Greenwood, Dalston, London*.—In case reported (No. 877) exposure to cold, and possible septic influences; and in another (not yet included), attack occurred in cold damp weather, but house was insanitary, and was the same one where the father and mother died of pneumonia, the latter immediately after former.
20. *H. Stoman, Farnham, Surrey*.—Believes large number of cases in the district, during spring and summer of 1883, to have been epidemic and probably contagious. In nearly every case, east wind prevailing at time, with a peculiar atmospheric condition commonly termed "blight." At same time mostly increase in other affections, as tonsillitis, whitlows, bronchitis, dyspepsia. Thinks strong ground for belief that some epidemic influence at work; for together with the pneumonia there occurred at same time many cases of acute tonsillitis, with decided constitutional symptoms, diphtheria, (at first very malignant), and a form of diarrhoea in which the symptoms were decidedly of a typhoid character and marked by asthenia. In no case could cause be found in defective drainage, milk supply, water supply or locality, the only cause common to all being the prevalence of east wind and the atmospheric "blight" above mentioned.
21. *P. Caldwell Smith, Motherwell, Lanark*.—Cases distinctly referable to exposure to cold; and believes that the change in direction of wind from south-west (most prevalent) to the east, is in many cases the cause.

The foregoing statements include not only facts, but the interpretations of the observers, with a result which deserves some attention. For this inquiry has elicited from 9 independent observers in various districts the opinion (Nos 2, 3, 6, 7, 9, 11, 17, 18, 19) that pneumonia under some circumstances appears capable of transmission from one individual to another, an opinion which has been previously expressed by others, especially certain Continental authors (*vide* articles on Epidemics). Most of our contributors, however, give this opinion under reserve, for in every instance of "apparent contagion" there is generally present the alternative explanation of subjection to like external conditions. The most striking instance, and the one least capable of this latter explanation, is that given by No. 3, who records very circumstantially the introduction of pneumonia into a household in a manner that recalls the mode of dissemination of a specific infective disease. In the other cases the evidence of such etiology is less conclusive, since it is obvious that where

more than one case occurs in the same house, the cause may lie in the condition of the dwelling, rather than in the individuals. Five observers (Nos. 4, 5, 7, 9, 20) speak of epidemics of pneumonia, attributable to atmospheric or meteorological conditions, a class which might perhaps be best compared with influenza-epidemics, between which and pneumonia a relationship has long been pointed out. Again, pythogenic causes were believed to operate in the experience of three observers (Nos. 2, 8, 16), whilst a belief in the influence of insanitary surroundings is expressed most strongly by Nos. 9 and 13. Exposure to cold (and wet) is regarded by several as the explanation of cases they have observed, with the reservation in some statements that the individuals attacked were predisposed by fatigue, debility, or constitutional condition, to become the subjects of pneumonia when so exposed. The influence of injury in inducing an attack of pneumonia is mentioned by one (No. 18).

Eighty other returns have been received upon this special point, but with hardly an exception, no other cause than exposure to cold can be assigned by the writers to the cases reported upon by them in this inquiry.

### *General Conclusions.*

THE large body of facts here brought together point to certain conclusions which, while they cannot be said in any case to reach the level of demonstration, are based upon different degrees of evidence whose precise value will be variously estimated according to the prepossessions of individual readers. The Report, therefore, might be left to speak for itself without any intervention of ours. For the most part, no doubt, it will be best so to leave it. Yet there are certain features of the Returns—most of them alluded to in the remarks appended to the several Abstracts—to which special attention may be called. The reader has before him the details of all the cases reported, and the means of verifying, or otherwise, any statement which is here advanced.

It must be admitted, in the first place, that much of the information elicited is of negative rather than positive value.



We are unable to assert that the incidence of pneumonia is in harmony with that of any other acute or specific disease, while the fact that bronchial and catarrhal affections, speaking generally, are apt to concur with it, was known already. On the other hand, the evidence that such concurrence is by no means invariable; the frequency with which pneumonia attacks more than one member of a household at the same time; the fact that when it is unusually prevalent its mortality is exceptionally low, and the rarity of enteric fever in any near association with it are all observations of value. Similarly, the inquiry into sanitary conditions, although it may not satisfy preconceived ideas, seems to show, first, that defective house drainage and sewer gas poisoning may both cause and favour the spread of pneumonia; secondly, that the affection when of this origin is not of exceptional severity or high mortality.

In other respects, too, the Returns throw light upon the important subject of etiology in its relation to the course and termination of pneumonia. The high mortality of the disease in the intemperate is no new observation, but the facts before us would seem to show that alcoholic excess is not only an important factor in determining the issue of pneumonia, but that it is often of itself the actual exciting cause of the affection. It may be asserted, indeed, that pneumonia owning this origin is, at all ages, the most fatal form of the disease known to us.

Furthermore the Report seems to supply sufficient ground for believing that fatigue and mental depression—if they be not, in some instances, efficient causes of pneumonia—are certainly, next to alcoholic poisoning, the most unfavourable vital conditions with which to meet it. It is true that the actual number of examples of this sort is not large. But they are striking of their kind, exhibiting a very high mortality among persons whose ages and habits render them the least liable to succumb to such a disease. (Abstract XIV).

Insufficient food—a term, perhaps, of too indefinite significance—although exhibiting a high mortality is associated with fatal pneumonia in a smaller proportion than either alcoholic excess, mental depression, or bodily fatigue.

As regards infectious pneumonia it will suffice to refer to Abstract XIV., and the Note that follows it, as well as to the

articles on British and Foreign epidemics. That pneumonia is sometimes conveyed from person to person must, we think, be admitted, notwithstanding that some of the examples quoted to that effect are capable of other explanation. Insanitary conditions—in which, probably, ill ventilation as well as defective drainage ought to be included—appear to favour such conveyance, and it would seem necessary that the intercourse should be intimate and prolonged like that of patient and nurse, or of bed-fellows. In such circumstances infectious pneumonia, we think, must be admitted as a reality, a rare characteristic of the disease of which we are not, at present, in a position to offer an explanation. Yet while admitting as much, it must be affirmed, at the same time, that pneumonia, as we commonly see it, has no infectious character.

In thus subtracting from the gross total the drunken, the overworked, the mentally depressed, and the underfed, we withdraw in much larger proportion a number that die.\* It does not appear, however, that by any further reduction (excepting, indeed, that of aged persons and perhaps infants), we can still further reduce the *rate* of mortality out of proportion to the number of individuals.

Various kinds of noxious exposure appear, as has been seen, amongst the alleged causes of pneumonia, but the numbers are inconsiderable and the mortality low.

But along with circumstances of habit and personal condition, whether provocative of pneumonia or influencing its mortality, we have to consider previous illnesses. It can hardly escape notice how small is the list of these. Looking both before and after the disease the record of the illnesses of pneumonia patients (infantile diseases excluded) is mostly a blank.

\* That the rate of mortality is thus materially lessened may be shown in figures as follows :—

The total mortality of 1065 is 192, about 1 in 5½.

Subtract from these (1) 105 *intemperate*, of whom 45 died.

(2) 14 mentally or physically *depressed*, of whom 9 died.

(3) 22 *infectious*, of whom 5 died.

There remain 924 representatives of chill pneumonia and of pneumonia to which no cause is, or perhaps can be, assigned, and of these 133 died, or about 1 in 7.

We may go further, and subtract a fourth class, viz., the aged (*i.e.* 64 over 65 years), of whom 25 died. There remain 864, of whom 108 died, or 1 in 8.

It may be mentioned in particular that acute rheumatism, which has sometimes been likened to pneumonia, is a very rare antecedent. Our positive information under this heading relates in fact to only two points : one, the illnesses of childhood which precede pneumonia ; the other, the frequency, or otherwise, of anterior attacks, with reference to the question how far such attack either predisposes to a second, or renders the course and event of a second appreciably less favourable than it would otherwise be.

As regards children's diseases, scarlatina and measles claim almost equality in this respect (the numbers are precisely equal for series B and C), and it would appear\* that 1 child in  $2\frac{1}{2}$  has had one or other of these diseases. But little importance can be attached to this, for it must be borne in mind (1) that the specific fever is not necessarily *immediately* connected with the pneumonia, and (2) that a proportion of the cases referred to are presumably broncho-pneumonia.

In reference to antecedent pneumonia we may speak more positively. About 1 patient in every 9 or 10 has suffered pneumonia before, and this is true for all the series. Nor is this fact to be taken alone. It is pneumonia and pneumonia only that figures among the anterior illnesses of these patients. It seems safe to conclude that one attack predisposes to another. When, however, we come to consider the subjects of these second attacks, it does not appear that their prospects in regard to recovery are appreciably worse than others.

The same cannot be said of *third* attacks. These would seem to be very rare, and our numbers are therefore too small to be of much value. But of the 16 examples of the kind (representing one case in every 60) as many as 4 died.

But the etiology of pneumonia has to be considered with reference not only to the individual, but also to his family and inherited disposition. Is the liability to pneumonia inherited, or is there any other form of lung disease to be met with in excess in the direct family history of the patients taken in the aggregate? We think that both these questions must be

\* The actual number under this heading in the table of Abstract VII. are not comparable, it must be remembered, with the other numbers, inasmuch as they relate to children alone, that is, to only 176 cases.



answered in the negative. The inquiry, however, is a very difficult one as respects phthisis. While it can hardly be alleged, having regard to the general incidence of the disease among the community, that the proportion of family phthisis met with in our Returns (Abstract VI.) is in any notable excess, it may certainly be said that a strongly marked tubercular history or a history of acute phthisis in several members of the same family is very rarely encountered.

A word may be said as to various characters of pneumonia in reference to the situation of the lung inflammation. The implication of both lungs—which, practically speaking, is as much as to say of both bases—is an event not only very variable in its incidence throughout the three series, but its rate of mortality, always high, is very unequal. The mean duration of this form of attack is also longer than the others, and gradual termination is the general rule. For these and other reasons we incline to the opinion that a certain number of cases of ambiguous significance are included under this category, and that it is here mainly that examples of doubtful or erroneous diagnosis may occur. However this may be, it is certain that the parity of the several series in respect of the local seat of inflammation becomes more apparent when one-sided pneumonias are taken by themselves. Whether in numbers or in rate of mortality there is little to choose between the three series when thus reduced (Abstract IX.). The death-rate is likewise much diminished and it is much the same for the right as for the left side.

In regard to the apex\* as a seat of pneumonia, it is to be noticed, 1st, that this seat is as favourable for the patient as any other (in its duration and mortality, indeed, it is more favourable than most).

2nd. That no tendency is observable on the part of patients of phthisical family to exhibit pneumonia at that seat.

3rdly. That in the exceptional event of an apex pneumonia occurring in a patient of phthisical family such pneumonia shows no tendency to degenerate into phthisis, but undergoes resolution as quickly and completely as another. Thus the

\* As with the base so with the apex it is to be understood that the latter term signifies the upper region of the lung—say its upper third. The Returns supply no data for determining the precise locality of the inflammation.

doctrine of the exceptional gravity of apex pneumonia finds no support in the Returns.

There is another point which, equally with the one just noted, may demand notice inasmuch as it conflicts with some accepted definitions of acute pneumonia. The proportion of sudden to gradual subsidence fails to bear out the statement that sudden remission of fever is the rule of the disease. The only law that obtains in this regard is this: that when the pneumonic pyrexia has lasted over 8 days its sudden subsidence is improbable, and that sudden subsidence after 10 days is very rare.

There are other matters of interest and importance which need not be rehearsed here, inasmuch as they are clearly set forth in the several Abstracts. Such are the days of crisis and the days of death, the maintenance of one rate of mortality with the indication thereby afforded that the course and termination of the disease are but little influenced by drugs.

There are, of course, many other points of clinical interest which do not fall within the scope of this Report. These have reference to the degree and variations of pyrexia, urea discharge, the amount of prostration, and the correspondence or otherwise of the local with the general symptoms. It was never contemplated to set forth such details in tabular form. On the contrary, the inquiry was purposely restricted so as to exclude them. It must be added, unfortunately, that there are other questions on which we thought to gather information—such as the intervals of time elapsing between first invasion and the earliest physical signs, or again, between the subsidence of fever and complete resolution of the affected lung—but the replies are so uncertain and fragmentary that we hesitate to deal with them in any summary manner.

It remains for us to consider how far the facts which have been now enumerated help to settle the problem of the essential nature of pneumonia; to determine, that is to say, whether it is to be regarded as a pulmonary inflammation only, or whether it is more truly a disease of "constitutional" nature, of which the lung affection forms but a part.

Without venturing to assert that any definite or final conclu-

sion can as yet be announced, we would point to certain facts in regard to pneumonia, witnessed to by the Returns, which suffice, in our opinion, to justify the conclusions we propose to draw from them.

The facts are these: In a certain proportion of instances of pneumonia there is no evidence of chilling or exposure, whilst unhealthy surroundings, defective and improper food or drink, bodily and mental fatigue, play no inconsiderable part, indirectly if not directly, in their causation. Add to this, that in many instances there has been an absence of that acute and sudden onset of symptoms which we are wont to associate with inflammation. Rigor is often absent, and for a variable time preceding the acute pulmonary invasion there is a premonitory stage of illness, with symptoms of general malaise, gastro-intestinal or nervous disorder. Again, pneumonia may occur in epidemic form when all the conditions provocative of pectoral affections are absent, and when, with the exception of pneumonia, all such affections are as a fact absent.

The question of infection in its present stage is not one of which we can make use, but the pythogenic origin of pneumonia is beyond question; as is the fact that its constitutional symptoms may be quite out of proportion with the localized symptoms, and even that the phenomena of the pneumonic fever may show themselves with no lung implication whatever.

Here, as we would submit, are so many arguments inclining to the belief that acute pneumonia should be regarded not merely as a local inflammation, but also as dependent—now more, now less—upon some profound cause, the nature of which is known to us as little as is the essential change which underlies acute rheumatism.

Keeping clear of hypothesis, we believe that the following statement defines and comprehends the several forms of the affection which have to be recognised and find illustrations in the present Report.

1. Of pneumonia as a local affection, there are examples in plenty, especially in early life. Its onset is sudden, and due to some notable chill or exposure of the body; it has all the characters of acute inflammation, with a marked tendency to spontaneous recovery, and is largely dependent on certain



meteorological conditions which are productive also of other forms of lung inflammation.

2. Distinguished from these are examples of secondary pneumonia, such as arise in the course of many acute and specific affections, and which do not at present concern us.

3. In addition to these two well-recognised forms of the disease, clinical observation, we think, bids us recognise a third variety—a pneumonia due to causes not directly injurious to the lung, but operating through the blood or nervous system. It, too, is properly a secondary pneumonia, for it is but the signal and expression of anterior vital changes; yet, owing to the absence (or the apparent absence) of any other organic lesion, it is not so accounted. Although anatomically indistinguishable (so far as we know at present), it deserves separate recognition in virtue as well of its distinctive origin as of the fact that it has a variable rate of mortality, and a gravity which is not commensurate with the extent of lung tissue involved.

*The Sub-Committee is indebted for the preparation of this report to* DRS. OCTAVIUS STURGES *and* SIDNEY COUPLAND.

(Signed) OCTAVIUS STURGES, M.D.  
T. H. GREEN, M.D.  
J. BURNEY YEO, M.D.  
SIDNEY COUPLAND, M.D.  
F. A. MAHOMED, M.B.  
W. P. HERRINGHAM, M.B., *Secretary*  
*to the Committee.*

# NAMES OF REPORTERS IN ALPHABETICAL ORDER AND THE NUMBER OF CARDS RETURNED BY EACH.

Name.	No. of Cases.	Name.	No. of Cases.	Name.	No. of Cases.
Abbott, C. E. ....	1	Black, J. R. ....	3	Cohen, A. A. ....	2
Adams, A. M. ....	1	Black, W. J. ....	2	Cole, T. ....	1
Addy, B. ....	1	Blackman, G. H. ....	1	Collett, G. B. ....	3
Adkins, J. E. ....	1	Blair, J. ....	2	Collin, J. T. ....	2
Aikman, J. ....	4	Blake, G. F. ....	1	Colman, G. M. ....	1
Alderson, F. H. ....	2	Booth, J. M. ....	10	Coombs, C. P. ....	5
Alexander, J. ....	2	Boulter, H. B. ....	1	Coombs, S. W. ....	7
Alexander, W. ....	1	Bourke, G. D. ....	3	Cooper, F. W. ....	1
Allan, F. J. ....	3	Boutflower, A. ....	1	Copley, W. H. ....	2
Allan, G. R. ....	2	Bowen, E. ....	2	Corbett, T. ....	6
Allan, Jas. ....	4	Bowen, O. ....	5	Corke, C. A. ....	1
Allen, J. E. ....	2	Boyce, C. ....	10	Cosgrave, E. M. ....	1
Allen, W. H. ....	1	Brabazon, A. B. ....	1	Craig, D. W. ....	1
Alliott, A. J. ....	3	Brabazon, W. P. ....	2	Crawford, D. G. ....	2
Anderson, R. ....	1	Bradbury, J. B. ....	1	Crossman, E. ....	1
Angrar, W. ....	1	Bradley, D. ....	2	Crowe, G. W. ....	1
Armstrong, W. ....	11	Bradley, W. E. ....	1	Cunnington, C. W. ....	1
Atkinson, F. P. ....	1	Bridger, J. ....	3	Cureton, E. ....	1
Atkinson, J. P. ....	1	Brock, A. C. ....	1	Currie, A. S. ....	2
Atkinson, R. ....	1	Brook, W. F. ....	1	Currie, D. W. ....	2
		Broomhead, C. ....	4		
Bain, D. B. ....	1	Brown, G. A. ....	2	Dalton, C. G. ....	1
Baird, J. W. ....	1	Brown, Jas. ....	1	Daly, F. G. H. ....	2
Baker, A. de W. ....	2	Brown, Jno. ....	4	Daly, E. O. ....	2
Bampton, A. H. ....	2	Browne, H. L. ....	4	Darwin, G. H. ....	2
Barfoot, G. H. ....	1	Bruce, W. ....	2	Davidson, A. ....	1
Barker, H. M. ....	3	Buckell, W. R. ....	1	Davies, D. A. ....	5
Barnes, E. G. ....	2	Burlingham, D. C. ....	1	Davies, G. A. ....	2
Barnish, W. C. ....	1	Burnett, W. E. S. ....	1	Davies, W. R. D. ....	1
Barr, J. ....	4	Burroughs, T. J. ....	3	Davis, G. H. ....	2
Bates, T. ....	1	Burton, S. H. ....	2	Dawson, C. ....	1
Bates, W. R. ....	1	Bury, J. S. ....	2	Dempsey, A. ....	2
Batho, R. ....	2	Buxton, T. ....	1	Denby, T. C. ....	1
Batterbury, G. H. ....	1			Derme, H. ....	2
Batterbury, R. L. ....	9	Campbell, A. ....	1	Derme, T. V. E. ....	1
Beatson, G. ....	1	Campbell, W. M. ....	3	Dickson, G. C. ....	1
Beattie, G. W. ....	1	Cant, W. T. ....	1	Dix, W. F. ....	4
Behrendt, M. R. J. ....	12	Cardew, G. A. ....	2	Douglas, C. E. ....	3
Bellingham, J. ....	2	Carmichael, M. ....	1	Douglas, J. G. D. ....	1
Bernard, W. ....	5	Carter, D'A. B. ....	3	Douty, J. H. ....	1
Berridge, W. A. ....	1	Cartwright, J. P. ....	2	Drake, F. H. ....	1
Berry, W. ....	2	Cattle, C. H. ....	1	Druitt, L. ....	2
Bevan, J. ....	1	Chaldecott, C. W. ....	1	Drummond, E. ....	2
Biddle, C. ....	3	Chapman, G. ....	1		
Biggs, M. G. ....	4	Clendinnen, J. G. ....	7	Easby, W. ....	2
Birt, G. ....	2	Cleveland, W. F. ....	2	Eastes, T. ....	3
Biss, C. Y. ....	1	Clippingdale, S. D. ....	6	Eddowes, A. ....	2
Black, G. ....	2	Clunn, T. R. H. ....	5	Ede, C. ....	1

Name.	No. of Cases.	Name.	No. of Cases.	Name.	No. of Cases.
Edmond, G. ....	4	Harrison, J. A. W. ...	1	Lloyd, A. E. ....	2
Edwards, J. ....	2	Haynes, F. H. ....	1	Lloyd, J. J. ....	12
Erskine, R. ....	1	Haynes, S. ....	1	Lloyd, R. R. ....	1
Evershed, A. ....	1	Hemsted, H. ....	1	Lucas, C. ....	1
		Hex, H. ....	1	Lucas, H. ....	1
Farmer, C. ....	2	Hill, C. H. ....	1	Luscombe, W. E. ....	4
Farrar, J. ....	1	Hoar, C. E. ....	1	Lush, W. V. ....	1
Faulkner, J. T. ....	1	Hodson, C. F. ....	1	Lynch, J. R. ....	1
Fenn, E. ....	1	Holburton, H. N. ....	1		
Fiddian, A. P. ....	9	Hollis, W. A. ....	2	McAldowie, A. M. ...	2
Fielding, T. ....	1	Holmes, J. ....	1	McClure, T. M. ....	1
Finlay, W. A. ....	3	Hope, E. W. ....	2	Macdonald, A. ....	1
Finlayson, A. ....	1	Howlett, E. H. ....	3	Macdonald, K. N. ....	1
Firth, C. ....	2	Hubbard, H. W. ....	2	Macdonald, R. W. ....	3
Fisher, F. B. ....	1	Hudson, R. S. ....	2	Macfarlane, A. W. ...	2
Fisher, F. C. ....	2	Hull, E. G. ....	3	Mackay, W. B. ....	1
Fitzgerald, C. E. ....	1	Humphreys, F. W. ...	2	Mackenzie, D. J. ....	8
Fleming, A. J. ....	2	Humphry, L. ....	1	Mackenzie, J. A. ....	7
Forty, D. H. ....	3	Hunt, De V. ....	6	Mackenzie, J. I. ....	1
Fountaine, D. O. ....	2	Hunter, G. ....	1	Mackinlay, J. ....	2
Fox, E. L. ....	3	Hunter, W. L. ....	3	Mackintosh, M. ....	1
Fraser, G. R. ....	1	Hutton, H. R. ....	6	McLachlan, S. F. ....	1
Frew, W. ....	4			McLachlan, W. A. ....	3
Fry, J. F. ....	5	Illingworth, C. R. ....	1	MacLean, A. ....	1
		Irvin, J. H. ....	1	McLeod, D. ....	1
Garner, J. E. ....	2			McMahon, J. T. ....	2
Garstang, T. W. H. ...	8	Jackson, E. ....	5	Macphail, D. ....	3
Gentles, R. W. ....	1	Jessop, C. M. ....	2	Macpherson, A. ....	3
Gentles, T. L. ....	1	Johnson, C. J. B. ....	1	Mallett, F. B. ....	4
George, J. T. ....	1	Johnson, S. ....	1	Malley, A. C. ....	5
Gibson, G. A. ....	2	Johnston, J. ....	5	March, H. C. ....	4
Gooch, J. W. ....	4	Joll, Boyd ....	1	Marsh, D. E. B. ....	1
Goyder, D. ....	1	Jones, C. M. ....	1	Marshall, J. S. ....	1
Graham, A. R. ....	1	Jones, Evan ....	1	Marshall, L. W. ....	1
Graham, T. F. ....	1	Jones, T. E. ....	4	Martin, Jas. ....	2
Gray, R. ....	1	Jones, W. D. ....	1	Martin, J. M. H. ....	7
Green, W. E. ....	6	Jones, W. F. ....	1	Martin, R. J. ....	1
Greenwood, M. ....	1	Jordan, F. W. ....	1	Martin, Theo. ....	1
Griffin, J. ....	1	Jordison, C. ....	1	Martin, T. ....	5
Griffith, S. ....	2	Joseph, G. W. ....	1	Mason, W. H. ....	1
Grosholz, F. H. V. ...	1	Joseph, J. F. ....	1	Matthews, J. ....	1
Grosvenor, A. V. ....	2	Joynes, F. J. ....	1	Mears, R. ....	2
Grove, W. R. ....	2			Meredith, J. ....	5
		Ker, H. R. ....	10	Mersiter, M. ....	3
Hadley, G. P. ....	1	Kerr, J. G. D. ....	1	Miall, P. ....	2
Haining, W. ....	1	Kershaw, H. ....	2	Michie, W. A. ....	2
Hall, T. L. ....	1	Kirk, R. ....	1	Mickle, W. J. ....	1
Hallowes, A. ....	1			Millard, W. W. ....	1
Hamilton, A. ....	2	Laing, H. W. ....	1	Miller, J. W. ....	1
Hamilton, H. ....	3	Lamb, W. ....	3	Millican, K. ....	1
Hamilton, Jas. ....	1	Lane, J. W. ....	12	Moir, J. W. ....	2
Hamilton, J. B. ....	1	Largrove, C. ....	1	Moore, J. W. ....	1
Handford, H. ....	1	Leachman, A. W. ....	4	Moore, N. ....	1
Hardey, E. T. ....	1	Le Grand, W. J. ....	2	More, J. ....	2
Hargreaves, M. K. ...	2	Lewis, C. G. M. ....	1	Morgan, T. ....	1
Harle, E. ....	1	Lilley, G. H. ....	2	Morison, B. G. ....	2
Harman, W. H. ....	1	Lillies, H. ....	1	Morris, W. ....	1
Harman, W. M. ....	1	Lipscomb, J. T. W. ...	1	Morris, W. J. ....	1
Harmer, W. M. ....	1	Lithgow, T. G. ....	1	Morshead, P. W. ....	4
Harrison, C. ....	4	Littlejohn, S. G. ....	5	Mortimer, W. ....	1



Name.	No. of Cases.	Name.	No. of Cases.	Name.	No. of Cases.
Moseley, W. A. ....	1	Reid, G. ....	1	Taylor, H. C. ....	2
Mullaly, W. J. ....	2	Reid, Jas. ....	3	Taylor, H. S. ....	1
Mulligan, J. W. ....	1	Reid, Jno. ....	4	Taylor, J. ....	2
Mulvany, J. ....	2	Renshaw, C. G. ....	4	Taylor, S. ....	1
Mumby, B. H. ....	1	Renshaw, H. S. ....	4	Terry, H. G. ....	3
Mumford, W. ....	1	Rice, R. ....	1	Thistle, F. T. ....	1
Munn, J. ....	4	Rich, A. C. ....	1	Thompson, H. G. ....	2
Murphy, T. C. ....	1	Robinson, A. H. ....	1	Thomson, E. ....	1
		Robinson, C. H. ....	1	Thomson, M. ....	1
Napier, A. ....	1	Robinson, T. ....	4	Thursfield, P. W. ....	4
Napier, A. D. L. ....	8	Robson, A. W. M. ....	4	Tomkins, A. W. ....	1
Neal, Jas. ....	1	Ronaldson, J. B. ....	1	Tylecote, J. H. ....	1
Neal, J. B. ....	2	Ronaldson, T. R. ....	3	Tylecote, E. T. ....	1
Neil, J. ....	3	Ross, R. ....	3	Tyson, W. J. ....	1
Nesfield, S. ....	1	Rossiter, G. F. ....	1		
Newman, T. W. ....	1	Routh, A. ....	1	Underhill, F. T. ....	2
Nurstead, C. V. ....	1	Russell, W. ....	2	Underhill, J. E. ....	5
Odell, W. ....	1	Salter, F. ....	1	Vaughan, W. S. W. ....	2
Ollerby, R. T. ....	2	Sanctuary, T. ....	1		
Ollerhead, T. J. ....	2	Schofield, G. T. ....	3	Wake, E. G. ....	1
O'Neil, H. ....	1	Scott, C. M. ....	1	Walford, W. G. ....	1
O'Neil, S. L. ....	2	Scott, E. S. ....	8	Walker, A. D. ....	1
Owen, S. H. ....	1	Scott, R. J. H. ....	1	Walker, E. G. A. ....	3
Owens, C. A. ....	1	Scougal, E. F. ....	5	Walker, F. ....	3
		Sellar, T. A. ....	1	Wallace, T. ....	1
Packer, W. H. ....	1	Sellers, W. B. ....	1	Walter, C. C. ....	2
Paget, W. S. ....	1	Sellers, W., jun. ....	2	Walters, J. ....	2
Palin, H. V. ....	1	Sells, H. T. ....	1	Ward, J. L. W. ....	2
Palmer, F. S. ....	2	Sergeant, W. R. ....	4	Warren, S. ....	1
Palmer, J. J. ....	1	Shaw, W. ....	2	Webb, V. G. ....	3
Park, R. ....	3	Shearer, G. ....	3	Webster, T. J. ....	4
Parkinson, C. H. ....	5	Sheen, A. ....	1	West, J. G. U. ....	1
Parsons, C. ....	1	Shelly, C. E. ....	7	Whitaker, S. H. ....	2
Parsons, F. W. ....	1	Sheppard, G. A. ....	1	White, J. ....	2
Parsons, H. ....	1	Sheppard, W. D. ....	8	White, W. ....	14
Parsons, T. E. ....	1	Sinclair, R. ....	6	Whittle, Glyn ....	1
Pearse, T. F. ....	7	Sinclair, W. ....	2	Whitwell, G. G. ....	2
Peart, R. ....	1	Slimmer, E. ....	7	Whitworth, W. ....	2
Pennington, T. R. ....	1	Sloman, H. ....	9	Wigmore, J. ....	2
Phillips, C. H. ....	1	Sloman, S. G. ....	1	Wilks, G. ....	1
Phillips, G. A. ....	2	Smith, D. T. ....	1	Williams, D. M. ....	7
Phillips, L. ....	1	Smith, J. E. ....	1	Williams, E. ....	2
Pilcher, W. J. ....	1	Smith, T. C. ....	2	Williams, Evan. ....	2
Plimmer, H. G. ....	1	Smith, R. ....	1	Williams, M. M. ....	2
Powell, G. D. ....	1	Smith, S. C. ....	1	Williams, Owen. ....	1
Power, G. E. ....	1	Smith, T. S. ....	1	Williams, W. E. ....	1
		Smith, W. G. ....	1	Williams, W. H. ....	6
Quirke, J. ....	1	Sneddon, W. ....	1	Wilson, J. O. ....	1
		Spence, W. J. ....	1	Wilson, S. T. ....	4
Rae, G. A. ....	1	Stanley, W. H. R. ....	1	Winter-Fisher, S. ....	1
Rand, J. ....	1	Stawell, J. C. ....	2	Wolfenden, J. W. ....	3
Ranking, J. E. ....	1	Stewart, A. ....	4	Wollaston, T. J. ....	1
Ransome, A. ....	3	Stevens, G. W. ....	1	Wood, E. J. ....	1
Rawdon, H. G. ....	1	Stone, H. S. ....	3	Wright, W. H. ....	1
Raven, T. F. ....	29	Stokes, R. L. ....	1	Wright, W. M. A. ....	1
Rayner, A. C. ....	2	Stuart, J. A. E. ....	6	Wynne, J. H. ....	1
Reckitt, E. B. ....	2	Stuart, H. O. ....	1		
Redwood, T. H. ....	1	Sutherland, A. ....	2	Yarr, M. T. ....	1
Reid, D. J. ....	1	Sultan, F. ....	1	Young, F. T. ....	1

# INDEX.

---

	PAGE
Abstainers, Number and Mortality of ...	32
Age with reference to Mortality ...	28
Alcohol, Pneumonia ascribed to ...	58, 59
Analysis of Foot Notes ...	57
Anomalous Symptoms ...	57
Anxiety preceding Pneumonia ...	59
Apex Pneumonia (note, p. 46) ...	40, 50
Bibliography, Epidemics :—	
British ...	9
Foreign ...	25
Bronchitis with Pneumonia ...	33
Bronchitis in Family History ...	39
Bronchitis in Previous History ...	40
Cases, Number and Distribution of ...	28
Chronic Pneumonia, Sequel of Pneumonia ...	53
Conclusions, General ...	64
Cough, Sequela of Pneumonia ...	53
Crisis, Days of ...	47, 48
Death, Days of ...	52
Debility, Sequela of Pneumonia ...	53
Diphtheria with Pneumonia ...	33
Double Pneumonia ...	50
Duration of Fever ...	47
Empyœma, Sequela of Pneumonia ...	53
Enteric Fever with Pneumonia ...	33
Epidemics, British ...	5
Epidemics, Foreign ...	25
Erysipelas, with Pneumonia ...	33
Etiology, Note on ...	60
Etiology, other than Chill or Exposure ...	58
Family History ...	39
Fatigue preceding Pneumonia ...	59
Fever, Duration of (note, p. 48) ...	47
Food, Sufficient and Insufficient ...	33

	PAGE
Habits ... ..	32
Herpes with Pneumonia...	33
Hospitals, Returns of ... ..	(note) 51
Infection, Pneumonia ascribed to ... ..	58
Injury, Pneumonia from ... ..	58
Intemperate, Mortality of ... ..	33
Locality and Climatic Conditions ... ..	36
Lung, Part of, Affected ... ..	45
Mania, Sequel of Pneumonia ... ..	53
Mortality according to Season ... ..	28
Mortality at different Ages ... ..	32
Mortality, Rate of ... ..	49
Mortality, London Hospitals ... ..	(note) 51
Nervous Origin of Pneumonia ... ..	58, 59
Observers and Localities ... ..	29
Phthisis, Family History of ... ..	39
Phthisis, Sequela of Pneumonia ... ..	54
Pleurisy, Sequela of Pneumonia ... ..	53
Premonitory Symptoms ... ..	42
Prevalence of Pneumonia ... ..	34
Previous Illnesses ... ..	40
Pythogenic Origin of ... ..	58, 59
Rheumatic Fever with Pneumonia ... ..	33
Rheumatic Fever in Previous History ... ..	41
Rigor, Present or Absent ... ..	44
Sanitary States of Houses and Districts ... ..	35
Sequelæ ... ..	53
Sex, Age, and Mortality ... ..	28
Sex in Pneumonia associated with Alcohol ... ..	60
Subsidence of Fever ... ..	47
Termination of Fever ... ..	49
Tonsillitis with Pneumonia ... ..	33
Treatment ... ..	54
Wind, Direction of ... ..	37



# APPENDIX.

## TABLES OF THE PNEUMONIA RETURNS.

It is necessary to explain the following tables by a few words of introduction, without which they may be difficult to understand.  
The card issued upon acute pneumonia ran as follows:—

No. 1.

ACUTE PNEUMONIA.—With regard to (1) its epidemic prevalence, (2) its communicability, (3) its association with other prevalent diseases and with defective sanitary conditions, (4) its symptoms, duration and result, (5) its treatment.

Observer's Name .....  
Address .....  
Date of last obs. ....  
(Reply where possible by erasing words on card.)  
Initials of patient. M. or F. Age.  
Married. Single. Widowed.  
Occupation.  
Temperate. Intemperate. Total Abstainer.  
Food—sufficient, insufficient.  
Place of residence.  
Locality—high, low, damp, dry, exposed, confined.  
Prevailing wind at onset of attack.  
Atmospheric condition—dry, damp, wet, cold, hot,  
mild, changeable, sun or clouds.

	In same House.	In District.
Number of cases of pneumonia ..		
any kind of fever*....		
catarrhal fever .....		
tonsillitis.....		
herpes .....		
bronchial catarrh ....		
crysipelas.....		

State number of each disease under care of observer.

\* Nature of prevalent fever.

Of the replies to these questions the greater number are arranged in a tabulated form in the following pages. The tables consist of eighteen columns.

1. contains the name and qualification of the observer.  
2. The sex.  
3. The age of the patient.  
4. contains the answers to the questions as to the amount of alcohol drunk, and of food taken by him.  
5, 6 and 7, give the concurrent illnesses under the care of the observer, the first containing the names of the diseases, the second the number of cases in the same house, the third the number in the surrounding district. These columns are enclosed by thick black lines.  
8 describes the sanitary condition, first of the house, second of the surrounding district.  
9 relates the cases of lung disease that have occurred in the patient's family.  
10 details the illnesses which he has himself previously undergone. The diseases of infancy have as a rule been omitted, and are only inserted where the youth of the patient or some other circumstance allowed the supposition that they might have affected the health at the time of the reported attack of pneumonia. Previous attacks of pneumonia have been printed in italics.

It remains to explain the abbreviations used. These are almost all to be at once understood referring to the card-form above. For the sake of clearness some are given below together with the word they represent.

y.=year; m.=month; w.=week; d.=day, or  
half of; r.=rigor; F.=father; M.=mother; B.=  
brother; S.=sister, U.=uncle; A.=aunt; R.=  
recovery; D.=death; +=many or prevalent;  
= information defective. Words put in in-

Sanitary condition of house—Good, bad, indifferent.  
" " district — Good, bad, indif-  
ferent.  
Family history of lung disease.  
Previous illnesses of patient, with dates,  
Attack preceded by rigors. Date.  
Pronomitory symptoms.  
Date of onset of attack.  
Part of lungs affected. R. base, apex.  
L. base, apex.  
Expectoration—Blood, rusty, white, none.  
Fever—Severe, moderate, mild.  
Highest range of temperature.  
Duration of fever.  
Termination of fever—Sudden, by gradual sub-  
sidence.  
Duration of physical signs.  
Result.  
Remarks on any special feature of case.  
Sequelæ.  
Plan of treatment.  
How long has patient been under care of observer?

(This card as soon as filled up to be returned to  
Secretary of the Local Sub-Committee).

COL.

- enumerates the premonitory symptoms which preceded the present attack.
- answers the question as to the mode of onset, stating whether the pneumonia was ushered in by a rigor or no.
- gives the part of the lungs which was affected.
- groups together the characters of the pyrexia describing, first its degree, second the number of days during which the temperature was raised, third the manner of its subsidence, whether sudden or gradual.
- states the number of days during which the physical signs of the disease could be detected.
- gives the result.
- the sequelæ.
- contains numbers referring to footnotes at the bottom of the page. These notes are in the words of the observer of the case, and are of great importance. They have been for the most part selected as illustrating either the cause of the attack, or unusual symptoms in its course, or important points in its history, or the mode of death.

verted commas indicate that for this statement the observer declares himself to be dependent upon the patient's account. Cases which occurred at the same time in the same house are enclosed in a bracket, which is placed in column 18.

Observer's Name.	Sex.	Age.	Temperate or other-wise. Food.	Concurrent cases of illness in	Same house.	Same district.	Sanitary state of house " " of district	Family history of lung disease.	Previous illnesses of patient.	Premontory symptoms.	Mode of onset.	Part of lungs affected.	Character Duration Termination of fever.	Duration of physical signs.	Result.	Sequelae.	Note.
1 T. Hall M.D.	M.	34	Temp. Suff.	Pneum. Rötheln. Tonsill. Bron. Cat. Erysip.		2 +	Ind. Ind.	0	Pneum. L., 3 y. ago.	Vomiting, pain in chest.	r.	L. base.	Mod. 7 d. Grad.	9 d.	R.	0	
2 G. A. Brown, M.R.C.S.	M.	55	Temp. Suff.	Pneum. Rötheln. Catarrh. Tonsill. Bron. Cat.		2 5 2 7	Good. Ind.	0	0	Fever, chest pain.	r.	Both bases.	Sev. 9 d. Grad.	28 d.	R.	Still slight solidity L. base. Debility.	
3 W. D. Sheppard, L.R.C.P.	M.	52	Temp. Suff.	Pneum. Scarlat. Tonsill. Erysip.		2 2 1 1	Ind. Ind.	0	0	Catarrh.	r.	R. base.	Sev. 8 d. Grad.	14 d.	R.	0	
4 " "	M.	69	Temp. Suff.	Pneum. Scarlat. Tonsill. Bron. Cat.		1 3 2 +	Ind. Ind.	F. } d. "de- 2 S } cline."	Asthma, 14 y. ago.	Apoplectic fit.		R. whole	Mod. till D.	till D. 7th d.	D.		
5 Chas. Morgan Jones, M.R.C.S.	F.	20	Temp.? Suff.	Pneum.	1	1	Ind. Ind.	—	0	0	r.	Both bases.	Sev. 5 d. Grad.	10 d.	R.	0	
6 J. Farrant Fry, L.R.C.P.	F.	39	Tot. abs. Suff.	Pneum. Tonsill. Bron. Cat.	1	+	Good. Good.	0	Pneum., 5 y. ago.	Abdominal pain.	0	R. apex. L. whole	Mod. till D.	till D. 9th d.	D.		
7 Evan Jones, M.R.C.S.	F.	24	Temp. Suff.	Pneum. Scarlat. Catarrh. Tonsill. Bron. Cat. Erysip.		6 20 12 30 12 2	Ind. Ind.	0	Measles, 3 y. ago.	General malaise.	r.	L. base.	Sev. 7 d. Grad.	28 d.	R.	0	
8 Arthur Ransome, M.D.	F.	20	Temp. Suff.	Pneum. Bron. Cat.	1	5 +	Good. Good.	M. asthma and emphysema.	0	Vomiting.	r.	Both bases.	Sev. 6 d. Sud.		R.	0	1
							Ind.	0	Pneum. 3 y. ago.		r.	L. base.	Sev. 21 d.		R.	0	2

11 A. P. Fiddian, M.B.	F.	28	Temp. Suff.	Pneum. Diphth. Tonsill. Bron. Cat	1	1 5 2 4	Ind. Bad.	0	0	Pain in side.	r.	L. apex. 19 d. Grad.	5 d. Sud.	40 d.	R.	0	}
12 "	F.	60	Temp. Suff.	"	"	"	"	0	0		—	L. base. till D.	Mild till D.	till D.	D. 7th d.		
13 "	F.	3	Temp. Suff.	Tonsill. Scarlat. Pneum.	+	few	Ind. Ind.	S. bronchitis. G. F. chr. bron.	0	Vomiting, purging.	0	L. base. 10 d. Sud.	Mod. 10 d. Sud.	12 d.	R.	0	
14 "	M.	7	Tot. abs. Suff.	Pneum.	1	1	Ind. Good.	—	Diphth., 2 y. ago.	Vomiting.	r.	L. base. 9 d. Sud.	Ser. 9 d. Sud.	11 d.	R.	0	
15 "	M.	17	Tot. abs. Suff.	Pneum. Measles. Bron. Cat.	2 8 3	2 3	Good. Good.	M. pneum. M's. S. d. phth. Bs. & Ss. bron	0	Headache, pain in side.	0	R. apex. 3 d. Sud.	Mod. 3 d. Sud.	3 d.	R.	0	Tonsill. on 10th d.
16 G. A. Davies, L.R.C.P.	M.	32	Temp. Suff.	Pneum. Tonsill. Erysip.	2 2 1	2 2 1	Good. Good.	0	—	Stiffness, pain in side, chil- liness, some d.	r.	L. base. 13 d. Grad.	Mod. 13 d. Grad.	16 d.	R.		
17 K. Millican, L.R.C.P.	F.	21	Temp.? Insuff.	Pneum.	1	1	Ind. Ind.	M. d. pneu- monic phth. All Bs. and Ss. took phthisical.	0	0	r.	Both bases.	Mod. 7 d. Sud.	17 d.	R.	0	
18 T. W. Thursfield, M.D.	F.	32	Temp. Suff.	Pneum. Tonsill. Herpes. Bron. Cat. Erysip.	2 3 1 +	2 3 1 2	Good. Good.	M. asthma.	Pneum. 6 y. ago.	Catarrh 1 w.	r.	Both bases.	Ser. 5 d. Sud.	over 14 d.	R.	—	
19 C. Lucas, M.R.C.S.	M.	64	Temp. Suff.	Pneum. Catarrh. Tonsill. Herpes.	1 6 1 4	1 6 1 4	Good. Ind.	0	0	Vomiting headache, pains in trunk and limbs.	r.	Both bases.	Ser. 21 d. Sud.	55 d.	R.	0	5

1. S. attacked similarly 1 w. later. 2. Fever terminated with free epistaxis, diarrhoea, and secretion of milk which had been almost suppressed for several d. 3. Profuse epistaxis on 3rd d., with a fall of T. which lasted 2 d.; aborted 33rd d.; foetus in 4th m. 4. Came up from country to nurse case 11, her daughter; was overpowered by effluvia of her breath and stools; fell ill after a few d. with same disease. 5. Acute jaundice on 4th d.



Observer's Name.	Sex.	Age.	Temperate or otherwise. Food.	Concurrent cases of illness in	Same house.	Same district.	Sanitary state of house " " of district	Family history of lung disease.	Previous illnesses of patient.	Premontory symptoms.	Mode of onset.	Part of lungs affected.	Character of fever. Duration Termination	Duration of physical signs.	Result.	Sequelæ.	Note.
20 Herbert Lucas, M.R.C.S.	M.	15	Temp. Suff.	Scarlat.	1	1	Good. Good.	Phthisis.	Pneum. some y. ago.	Pain L. shoulder, fever.	r.	L. whole	Sev. 28 d. Grad.	28 d.	R.	0	1
21 Ezra Harle, L.R.C.P.	M.	38	Temp. Suff.	Pneum. Scarlat. Catarrh. Tonsill. Herpes. Bron. Cat. Erysip.	2 12 3 1 1 4 3	2	Good. Bad.	F. chr. bron.	0	Pain in back	0	L. base.	Sev. 9 d. Sud.	15 d.	R.	0	
22 W. R. Grove, M.D.	F.	11	Tot. abs. Suff.	Pneum.	1	1	Ind. Ind.	0	Skin disease, delicate.	—	r.	R. whole	Mod. 5 d. Grad.	7 d.	R.	0	
23 "	F.	57	Temp. Suff.	Pneum.	1	1	Ind. Ind.	0	Congestion R. lung.	0	r.	R. base. L. whole	Sev. 10 d. Grad.	16 d.	R.	Some soli- dification.	
24 W. Easby, M.D.	M.	8	Temp. Suff.	Tonsill. Scarlat. Bron. Cat.	2 1 +	2	Ind. Ind.	F. } d. phth. M. }	Acute bron.	Langnor, de- bility.	0	R. base.	Sev. 5 d. Grad.	5 d.	R.	0	2
25 "	M.	13	Temp. Suff.	Scarlat.	+	+	Ind. Ind.	0	Acute rheum. 6 y. ago. Ac. bron.	Langnor.	r.	L. base.	Sev. 10 d. Grad.	7 d.	R.	0	
26 W. H. Copley, L.R.C.P.	M.	51	Temp. Suff.	Measles.	+	+	Ind. Ind.	0	—	Pain inside, cough, fever	r.	R. base.	Sev. 10 d. Sud.	till D.	P. 19th d		3
27 J. More, M.D.	M.	45	Temp. Suff.	Pneum. Bron. Cat. Erysip.	4 4 2	4	Ind. Ind.	0	Pneum. 3 y. ago.	0	r.	R. base. L. whole	Sev. 10 d. Grad.	16 d.	R.	0	
28 Alexander Dempsey, M.D.	F.	6	Tot. abs. Suff.	Measles.	+	+	Ind. Good.	0	Worms.	Headache, cough, fever, pain in side.	r.	L. base.	Sev. 8 d. Grad.	13 d.	R.	0	4

	30 W. Bernard, M.D.	31 J. More, M.D.	32 J. P. Atkinson, M.D.	33 J. B. Bradbury, M.D.	34 J. Bridger, M.R.C.S.	35       "	36 E. C. Bury, M.D.	37 Hugh R. Ker, F.R.C.S.	38 W. A. Berridge, M.R.C.S.	39 A. C. Brock, L.R.C.P.	40 Chas. Ede, M.R.C.S.
Sex	F.	M.	M.	M.	F.	M.	M.	M.	M.	M.	M.
Age	13	37	22	30	34	71	36	48	24	32	36
Ref.	Tot. abs. Suff.	Temp. Suff.	Temp. Suff.	Temp. Suff.	Temp. Suff.	Temp. Insuff.	Temp. Suff.	Intemp. Insuff.	Temp. Suff.	Intemp. Suff.	Intemp. Suff.
Local	Catarrh.	—	—	Herpes. Bron. Cat.	Pneum. Bron. Cat.	Pneum. Bron. Cat.	Pneum.	—	Pneum. Bron. Cat.	—	Tonsill. Bron. Cat.
General	7	—	—	1 2	2 5	2 4	2	2	2 2	Ind. Good.	1 1
Ind.	Good. Ind.	Ind. Good.	Good. Ind.	Good. Ind.	Good. Ind.	Ind. Ind.	Ind. Ind.	Bad. Ind.	Good. Good.	Ind. Good.	Ind. Good.
0	0	1 S. d. phth.	2 daught. d., strumous habit, bron.	0	0	S. d. consump- tion 29.	0	0	1 B. d. bron- cho-pneu- monia.	F. d. consump- tion.	0
Scrophulous ophthalmia often.	Scrophulous ophthalmia often.	Continued fever, 6 w. previous.	—	Dyspepsia for 3 y.	0	Chr. bronch. many y. Pneum. 3 y. ago.	0	0	Pleurisy L. 1 y. ago.	0	Disease of bone of arm 1 y. ago.
Pain in side	Headache, malaise.	0	Catarrh.	0	Pain in side.	0	0	Lumbago.	Shivering, headache, sickness.	Malaise a few d.	Anorexia.
R. base. L. apex.	R. base. L. apex.	R. base.	Both bases.	L. base.	L. whole	R. base.	R. base.	R. base. L. whole	L. whole	R. base.	R. base.
Sev. 14 d. Grad.	Sev. 14 d. Grad.	Mild 28 d. Grad.	Mod. 12 d. Grad.	Mod. 7 d. Sud.	Mod. 12 d. Grad.	Sev. till D. 5th d.	Mod. 10 d. Grad.	Sev. till D. 13th d.	Sev. 14 d. Grad.	Sev. 10 d. Grad.	Sev. 4 d. Sud.
10 d.	10 d.	35 d.	—	over 9 d.	21 d.	till D. 5th d.	23 d.	till D.	—	21 d.	10 d.
R.	R.	R.	R.	R.	R.	D. 5th d.	R.	D. 13th d.	D.	R.	R.
0	0	0	—	—	Pain in side.	—	0	—	Pneumo- hydro- thorax.	0	0

1. T. began to fall on 15th d. and fell grad. during 14 d. 2. Catarrhal pneumonia. 3. 5 d. from onset arthritis of knees and elbows (?septic); 1 w. later thrombosis of L. femoral vein. 4. Measles 1 m. later; M. took pneum. after patient's recovery. 5. T. not above 100. 6. Acute mania 11th day; measles intercurrent 14th d.; sent to Bethlem 34th d.; well in 4 m. 7. Got a second and third attack with severe hemoptysis; D. 10 m. after first.

Observer's Name.	Sex.	Age.	Temperate or otherwise. Food.	Concurrent cases of illness in	Same house.	Same district.	Sanitary state of house " " of district	Family history of lung disease.	Previous illnesses of patient.	Prenatal symptoms.	Mode of onset.	Part of lungs affected.	Character of fever. Duration of fever.	Duration of physical signs.	Result.	Sequelæ.	Note.
41 R. Anderson, L.R.C.P., Surg. Maj. A.M.D.	M.	35	Tot. abs. Suff.	Ague. Catarrh. Tonsill. Bron. Cat.	1 } 4 } 2 } 8 }	Barracks.	Good. Ind.	0	Syph. Rheum. twice Orethritis. Ague, twice, last 1 y. ago.	Bron. cat., fever, sweat- ing, foul tongue, tonsill.	r.	R. apex. L. whole	Sev. 5 d. Sud.	16 d.	R.	Cough. debility.	
42 J. G. D. Douglas, M.D.	M.	10	Tot. abs. Suff.	Catarrh. Bron. Cat.		2 +	Good. Ind.	Phthisis.	0	Chilliness, pains, mal- aise.	—	R. base.	Mod. 7 d. Grad.	14 d.	R.	0	
43 C. H. W. Parkin- son, M.R.C.S.	F.	13	Temp. Suff.	Bron. Cat.		+	Ind. Ind.	M's 25 } d. M's M } phth. 2 Bs pneum. 2 Ss " " (4 cases pneu. in 5 y.)	Angular cur- vature. Chron. cough. Valvular heart disease.	Pain in side, illness.	—	R. whole L. base.	Sev. 14 d. Grad.	30 d.	R.	—	
44 "	M.	64	Intemp. Suff.	Bron. Cat.		+	Ind. Ind.	?	?	Cough, pain in side, shivering.	—	Both bases.	Mod. 14 d. Grad.	27 d.	R.	—	1
45 "	F.	17	Temp. Suff.	Bron. Cat.		2	Bad. Ind.	M's. F. d. asth- ma 52. M's. S. d. croup. 1 S " "	0	—	r.	L. base.	Mod. 7 d. Grad.	22 d.	R.	0	
46 Harry Hex, M.R.C.S.	M.	32	Temp. Suff.	Pneum.		2	Ind. Ind.	0	0	Ill 12 d.	r.	R. base	Sev. 14 d.	15 d.	D.		
47 W. E. Green, M.R.C.S.	F.	5	Tot. abs. Suff.	Pneum. Scarlat. Erysip.		+	Good. Good.	0	Yearly bron. severe.	Shivering.	r.	R. base.	Sev. 9 d. Sud.	11 d.	R.	0	
48 T. Sanctuary, M.D.	M.	56	Intemp. Suff.	Tonsill. Bron. Cat.		2 3	Good. Ind.	M. d. bronch. at 45.	0	Headache.	r.	R. base.	Mod. 6 d. Grad.	10 d.	R.	0	
49 W. E. Green.	M.	47	Intemp.	Pneum.		2	Good	0	0	0	r.	L. base.	Mod.	20 d.	R.	0	





Observer's Name.	Sex.	Age.	Temperate or other-wise. Food.	Concurrent cases of illness in	Same house.	Same district.	Sanitary state of house " " of district	Family history of lung disease.	Previous illnesses of patient.	Premontory symptoms.	Mode of onset.	Part of lungs affected.	Character of fever. Duration	Duration of physical signs.	Result.	Sequelæ.	Note.
62 J. Bevan, M.B.	F.	29	Temp. Suff.	0			Good. Good.	Some have "delicate chests."	Acute rheum. 8 y. ago.	Headache, slight hemiplegia.	r.	Both bases.	Sev. till D.	till D.	D. 10th d		
63 J. W. Mulligan, M.D.	M.	34	Intermp. Suff.	Scarlat. Bron. Cat.		+	Good. Ind.	—	—	—	—	Both bases.	Sev. —	—	D.		1
64 O. E. B. Marsh, L.R.C.P.	F.	32	Temp. Suff.	Pneum. Catarrh. Tonsill. Bron. Cat.	1 1 1	4	Good Good.	Phthisis.	Pleurisy R. 5 y. ago.	Fever, cough.	r.	Both bases.	Sev. 18 d. Grad.	Over 42 d.	R.	—	2
65 G. A. Davies, L.R.C.P.	F.	29	Temp. Suff.	—			Good. Good.	G. F. d. phth. 65.	Never strong.	Chills, aches.	r.	L. base.	Sev. 8 d. Grad.	21 d.	R.	Bad cough.	
66 D. H. Forty, L.R.C.P.	M.	55	Temp. Suff.	0			Good. Good.	0	0	Shivering.	r.	R. base.	Mod. 7 d. Grad.	28 d.	R.	0	3
67 "	M.	44	Intermp. Suff.	Tonsill. Bron. Cat.		1 1	Ind.	F. d. "22 w. after taking cold" at 40.	0	Stomach disturbed.	r.	R. base.	Sev. till D.	till D.	D. 6th d.		
68 C. Parsons, M.D.	M.	31	Temp. Suff.	—			Ind. Good.	0	Delicate.	Catarrh, bilious vomiting, pain R. scapula.	0	Both bases.	Sev. 8 d. Grad.	21 d.	R.	0	
69 T. F. Raven, L.R.C.P.	F.	14	Tot. abs. Suff.	0			Good. Good.	F's U. d. phth. M's M. bronch.	Pertussis severely 7 y. ago.	0	r.	R. apex to base.	Sev. — Grad.	—	R.	Loss of hair.	4
70 "	M.	50	Intermp. Insuff.	Pneum. Measles. Catarrh. Tonsill. Pneum.	1 + +	+	Ind. Good.	M. asthma.	Asthma, bron. for 24 y.	Chest worse some w.	r.	L. whole	Mild. till D.	till D.	D.		5
71 "	M.	6	Tot. abs. Insuff.		2 +	1	Ind. Good.	M's 2 A. d. phth.	Chronic diarrhoea, weakly.	Diarrhoea 5 d. then sharp	r.	L. apex.	Mod. 18 d.	22 d.	R.	Caught pertussis.	

72	"	M.	30	Intemp.	Pneum. Tonsill. Herpes. Bron. Cat.	2 9 1 1	Good. Good. Good.	Cases of pneum. have occurred.	Ulcerated throat 1 y. ago	Frontal headache, fever.	r.	R. base.	Mild. 6 d. Grad.	12 d.	R.	0
73	"	M.	19	Temp. Suff.	Measles. Erysipelas.	+	Ind. Good.	0	0	Flying pains in limbs ad., felt well.	r.	L. base.	Mod. 34 d. Sud.	14 d.	R.	0
74	F. Sutton. M.R.C.S.	F.	17	Tot. abs. Insuff.	Pneum. Scarlat. Tonsill. Catarrh. Bron. Cat. Erysip.	3 2 2 1	bad. Ind.	2 s. 1 A. and others	Menorrhagia, 1 y. ago.	Sickness, headache, pain in limbs	—	Both bases.	Sev. 18 d. Grad.	42 d.	R.	—
75	W. Bruce, M.D.	M.	60	Slightly Intemp. Suff.	Enteric.	few	Good. Good.	0	0	Stitch, dysp- noea.	r.	R. whole	Sev. — Grad.	over 5 d.	R.	—
76	A. Finlayson. L.R.C.P.	M.	—	Temp. Suff.	0	Good. Good.	Good. Good.	0	0	0	r.	R. base.	Mod. till D.	till D. 6th d.	D.	7
77	R. Batho, M.D.	M.	21	Tot. abs. Suff.	Pneum. Bron. Cat.	3 3	Good. Bad.	—	—	B. loose, anorexia, thirst.	r.	Both bases.	Sev. 12 d. Grad.	12 d.	R.	—
78	"	M.	33	Intemp. Suff.	Bron. Cat.	3	Good. Bad.	F. } "d." M. } asthma."	Ague often. Bronchitis. Syphilis last y.	Agueish attacks, anorexia, furred tongue, P. cough.	r.	Both bases.	Mod. 8 d. Grad.	18 d.	R.	—
79	T. F. Raven, L.R.C.P.	F.	11	Tot. abs. Suff.	Pneum. Scarlat. Tonsill. Bron. Cat. Erysip. Measles	3 2 6 + 2 +	Good. Good.	M's F's family phth. F's family phth.	Asthma occa- sionally, measles at 7, pertussis at 6	Catarrh.	r.	R. apex.	Mod. 4 d. Sud.	5 d.	R.	—
80	"	M.	35	Intemp. Suff.	Tonsill. Bron. Cat. Measles.	6 3 +	Ind. Good.	0	0	Giddy and queer 2 d.	r.	L. base.	Mild. 7 d. Sud.	7 d.	R.	—
81	C. H. Hill, M.D.	M.	7	Tot. abs. Suff.	Pneum. Catarrh. Tonsill. Bron. Cat.	4 + + +	Ind. Ind.	0	Bronch. last y. tonsill. often.	Tonsill. fever.	0	R. base.	Mod. 7 d. Sud.	11 d.	R.	—

1. Had a drinking bout 3 d. 2. Diphtheria set in at beginning of 3rd w. 3. Sudden remission 7th d.; relapse and prolonged convalescence. 4. Fever for 10 d., remission 2 d., fever for 4 w., signs remained for 3 m., P. persistently high, copious discharge of pus and signs of abscess. After 3 y. some damage still remained. 5. Kidneys unsound. 6. Symptoms of blood poisoning, severe; menorrhagia during illness, nearly fatal. 7. D., with typhoid symptoms.



Observer's Name.	Sex.	Age.	Temperate or other-wise. Food.	Concurrent cases of illness in	Same house.	Same district.	Sanitary state of house " " of district	Family history of lung disease.	Previous illnesses of patient.	Prenatal symptoms.	Mode of onset.	Part of lungs affected.	Character of fever. Duration Termination	Duration of physical signs.	Result.	Sequelae.	Note.
82 B. G. Morison, M.B.	F.	42	Temp. Suff.	Ac. Rheum. Tonsill.		3 1	Fair. Good.	0	?	Pleurisy 14 d., just con- valescent.	—	Both bases.	Sev. till D.	till D.	D.		1
83 "	M.	20	Temp. Suff.	Enteric Pertussis.		2 2	? Ind.	—	Bronch. often, asthma long, pleurisy.	Diarrhea.	r.	R. base.	Sev. 6 d. Sud.	7 d.	R.	0	
84 J. Johnston, M.D. (Bolton).	F.	13	Tot. abs. Suff.	0			Good. Ind.	F. pneum. at 20.	Measles, scarlat., enteric, croup.	—	0	R. base.	Mod. — Grad.	—	R.	0	
85 "	M.	22	Temp. Suff.	Erysip.		2	Ind. Ind.	F. d. phth. at 42.	0	0	r.	R. base.	Sev. 7 d. Grad.	11 d.	R.	0	
86 F. B. Mallett, M.D.	M.	47	Temp. Suff.	0			Good. Good.	0	0	Lassitude, chilliness.		R. base.	Mod. 5 d. Grad.	10 d.	R.	0	
87 James Barr, L.R.C.P.	M.	39	Temp. Suff.	Pneum.		2	Good. Good.	0	0	0	r.	R. apex.	Mod. 9 d. Grad.	14 d.	R.	0	
88 C. Dawson, L.R.C.P.	F.	64	Tot. abs. Suff.	Measles. Tonsill. Bron. Cat.		+ 2 few.	Good. Ind.	0	Feverish attack few y. ago, gouty diath.	Pains, malaise.	r.	R. apex.	Sev. 9 d. Sud.	12 d.	R.	—	2
89 De Vere Hunt, L.R.C.P.	M.	22	Temp. Suff.	Tonsill.	1		Ind. Ind.	0	Pneum. 6 y. ago.	Malaise.	r.	R. base.	Mod. 7 d. Grad.	11 d.	R.	Debility.	
90 C. J. Renshaw, M.D.	M.	8	— Suff.	Pneum. Measles. Catarrh. Tonsill. Herpes. Bron. Cat. Erysip.		3 20 11 5 3 4 1	Ind. Ind.	0	Measles 3 y. ago.	Catarrhal fever.	r.	R. base.	Sev. 8 d. Grad.	5 d.	R.	0	

91 H. R. Hutton, M.B.	F.	14	— Insuff.	0	—	0	Measles.	Febr., anorexia, thirst.	L. base.	Nov. 13 d. Grad.	29 d.	R.	Diarrhoea.
92 "	F.	7	Temp. Suff.	0	—	—	Pertussis.	Headache, vomiting.	—	Mild. 7 d. Sud.	21 d.	R.	
93 "	M.	3½	— Insuff.	0	Ind. Ind.	1 other child d. bronchit 4 m.	Measles 2 y. ago.	Vomiting, fever, pain in abd.	R. apex.	Mod. 6 d. Sud.	19 d.	R.	Bronchitis. 3
94 "	M.	9	Tot. abs. Insuff.	0	—	F. d. phth.	0	—	r. R. apex.	Mod. 7 d. Grad.	28 d.	R.	0
95 "	M.	11	—	0	—	—	Measles.	Headache, vomiting, diarrhoea.	0 R. apex.	Mod. 7 d. Sud.	21 d.	R.	Otorrhoea.
96 "	M.	10	— Insuff.	0	Ind. Ind.	No phth.	0	Chilliness, lassitude, vomiting.	—	Mod. 9 d. Grad.	15 d.	R.	0
97 G. H. Darwin, M.D.	M.	16	Temp. Suff.	0	Ind. Good.	0	0	—	r. L. base.	Sev. 20 d.		R. part.	Pleurisy, emphysema, paracene- tesis. 4
98 "	M.	20	Intemp. Insuff.	Pneum. Scarlat. Erysip.	1 2 1	—	0	Langour, anorexia.	r. R. apex.	Mod. 9 d.	18 d.	R.	0 5
99 Judson S. Bury, M.D.	M.	5	Tot. abs. Suff.	—	Good. Good.	0	—	—	0 R. base.	Mod. 10 d. (?) Grad.	10 d.	R.	0 6
100 "	M.	2½	Tot. abs. Suff.	Pneum.	1	Good. Good.	0	Convulsions, scarlat., measles.	0 L. base.	Sev. 14 d. Grad.	21 d.	R.	0
101 W. C. Barnish, M.R.C.S.	M.	—	— Suff.	Bron. Cat.	2	Bad. Good.	0	—	r. L. base.	Mod. 10 d. Grad.	12 d.	R.	0

1. Came on in an otherwise normal puerperium; albuminuria; diarrhoea throughout case; D. by sudden collapse at crisis. 2. Physical signs latent till 8th d., then only over area size of half-a-crown. 3. Slight albuminuria. 4. Caused by injury to chest wall. 5. Wife d. pneum. 5 d. before onset; he slept with her and neglected his food. 6. Brothers; case 100 was seized on 13th d. of illness of case 99, when 99 was convalescing; they were in the same room and 100 was not exp. to cold.

Observer's Name.	Sex.	Age.	Temperate or otherwise. Food.	Concurrent cases of illness in	Same house.	Same district.	Sanitary state of house " " of district	Family history of lung disease.	Previous illnesses of patient.	Premontory symptoms.	Mode of onset.	Part of lungs affected.	Character of fever. Duration. Termination.	Duration of physical signs.	Result.	Sequelæ.	Note.
102 T. F. Raven, L.R.C.P.	M.	4½	— Suff.	Pneum. Tonsill. Bron. Cat. Measles. Erysip.	1 3 2 + +	1 3 2 + +	Good. Good.	M's B. d. phth. F's S. d. phth.	0	Catarrh and cough 1 m.	0	L. apex to base.	Sev. 7 d. Grad.	8 d.	R.	—	1
103 O. Williams, L.K.Q.C.P. (Rhosygaer).	M.	64	Temp. Suff.	Enteric. Erysip.	2 2	2 2	Good. Good.	—	Chr. bron.	Fever, dyspnoea, cough, anorexia.	r.	R. whole L. base.	Sev. 17 d. Grad.	44 d.	R.	—	
104 " "	M.	40	Temp. Suff.	Enteric. Erysip.	2 2	2 2	Ind. —	0	0	Fever, headache, foul tongue, dyspnoea.	r.	L. whole	Sev. — —	—	—	—	2
105 E. Williams, L.R.C.P. (Bala).	F.	33	Temp. Suff.	0			Ind. Good.	0	Catarrhs.	0	r.	R. base.	Mod. 7 d. Grad.	12 d.	R.	0	
106 Evan Williams, L.K.Q.C.P. (Llangefni).	M.	30	Temp. Suff.	Pneum. Pertussis. Tonsill. Erysip.	3 + 6 3	3 + 6 3	Ind. Ind.	F. d. bronch.	0	0	r.	R. base.	Sev. 10 d. Grad.	18 d.	R.	0	
107 D. M. Williams, L.K.Q.C.P. (Liverpool).	M.	6½	Temp. Insuff.	Measles.	+	+	Ind. Ind.	F. pneum. M. pleurisy.	Pertussis 2 y. ago.	Fever 3 d.	0	R. base.	Mod. 20 d. Grad.	27 d.	R.	0	
108 Jas. Edwards, L.R.C.P.	F.	23	Tot. abs. Suff.	Measles. Catarrh. Bron. Cat.	1 2 1	1 2 1	Good. Good.	1 S. pneum.	0	0	r.	Both bases.	Sev. 2 d. Sud.	—	R.	—	3
109 S. Griffiths, M.D.	F.	35	Temp. Suff.	0			Good. Good.	F. } d. phth. B. }	Rheum. fever.	Cough, stitch, thirst.	r.	Both bases.	Sev. till D.	till D. 8th d.	P.		
110 F. H. V. Gros- holz, M.K.Q.C.P.	M.	18	Tot. abs. Suff.	Bron. Cat.	1	1	Ind. Good.	0	0	Vomiting.	r.	R. base.	Sev. 10 d.	15 d.	R.	0	



L.R.C.P. (Bala)		Suif.			Bad.	at 22. 1 B. phth.	ago.		r.	R. base.	11 d. Grad.	
112	John Meredith, M.D.	28	M.	Tot. abs. Suif.	—	1 S. pneum. twice.	0	Unwell some d.	0	R. base.	Sev. 5 d. Sud.	0
113	"	32	M.	Temp. Suif.	Tonsill. Bron. Cat.	4 3	Hæmopt. or hæmatem. 5 y. ago.	Cough, diar- rhea some d.	0	Both bases.	Sev. 7 d. Grad.	—
114	J. Bellingham, M.R.C.S.	35	M.	Temp. Suif.	0	F. d. bronch. at 67.	Catarrh 2 y. ago.	Nausea.	0	R. base.	Sev. 10 d. Grad.	0
115	D. Bradley, L.R.C.P.	18	M.	Temp. Suif.	Scarlat. Enteric.	+	Scarlat 1 y. ago.	0	0	R. base.	Sev. 4 d. Sud.	0
116	T. Buxton, M.R.C.S.	23	M.	Temp. Suif.	Pneum. Catarrh. Tonsill. Erysip. Bron. Cat.	2 5 4 2 +	Bronch.	Pain in side.	0	R. base.	Sev. 5 d. Grad.	—
117	W. Macfie Camp- bell, M.D.	15	F.	Tot. abs. Suif.	Pneum. Measles. Catarrh. Tonsill. Herpes. Bron. Cat. Erysip.	1 3 8 3 1 7 1	Diphtheria 3 y. ago, measles 2 y. ago.	0	0	R. base.	Mod. 5 d. Grad.	—
118	J. E. Garner, M.D.	25	F.	Temp. Suif.	—	Good. Good.	0	Pain in sides and back.	0	Both bases.	Sev. 7 d. Sud.	0
119	J. M. H. Martin, M.D.	24	F.	Tot. abs. Suif.	0	Good. Ind.	Neuralgia 3 m. ago.	Chills, head- ache, vomit- ing.	0	R. base.	Sev. till D.	3
120	W. Sinclair, M.D.	33	M.	Temp. Suif.	Pneum. Enteric.	2 1	---	0	0	L. base.	Mod. 7 d. Sud.	0

1. Convulsion in place of rigor at onset. 2. On board ship; only 4 d. under care. 3. Chill during menses. 4. Offensive privy and pigsty; family suffers every June with diarrhœa; this case was in July. 5. D. by coma.

Observer's Name.	Sex.	Age.	Temperate or otherwise. Food.	Concurrent cases of illness in	Same house.	Same district.	Sanitary state of house " " of district	Family history of lung disease.	Previous illnesses of patient.	Premonitory symptoms.	Mode of onset.	Part of lungs affected.	Character of fever. Duration of physical signs.	Result.	Sequelae.	Note.
121 W. Sinclair, M.D.	M.	48	Temp. Suff.	Pneum. Enteric.		2 1	Good. Good.	F. d. pneum. at 66.	Variola 10 y. ago.	Cold & cough somed.	r.	Both bases.	Mod. 7 d. Sud.	R.	0	
122 G. W. Steeves, L.R.C.P.	M.	6	Tot.abs. Suff.	Pneum. Tonsill. Bron. Cat. Scarlat. Measles. Erysip.	1	1 2 2 5 2	Good. Ind.	M. chr. bron.	Pneum. 5 m. ago.	Headache, vomiting.	r.	L. base.	Sev. 12 d. Grad.	R.	Part of L. base still solid.	
123 J. W. Moore, M.D.	M.	36	Intemp. Suff.	Pneum. Enteric.	1	2	Ind. Ind.	0	0	Sweating, headache, pain in side.	r.	R. base.	Mod. 8 d. Sud.	R.	—	1
124 J. Wilmore, M.D.	M.	41	Temp. Suff.	Pneum. Herpes. Bron. Cat. Erysip.	2 1	8 3 20 4	Ind. Ind.	0	English cholera 5 y. ago.	Chilliness, cough, thirst, pain in side.	r.	L. base.	Mod. 7 d. Sud.	R.	0	}
125 "	F.	15	Tot.abs. Suff.	" "			" "		—	Sev. catarrh 4 d., general pains.	0	L. base.	Sev. 7 d. Grad.	R.	Pain in side, debility.	
126 T. Cole, M.D.	F.	29	Tot.abs. Suff.	0			Good. Good.	0	0	Pain in limbs and side.	r.	R. whole L. base.	Mod. 7 d. Sud.	R.	—	3
127 G. F. Rossiter, M.B.	M.	14	Temp. Suff.	Bron. Cat. Erysip.	1	2 1	Good. Good.	M. croup often when young.	Double pneum. this time last year.	Cough, anorexia, dreams.	r.	Both bases.	Mild ? Grad.	R.	0	4
128 J. P. Cartwright, M.R.C.S.	F.	47	Temp. Suff.	Enteric. Tonsill. Bron. Cat. Erysip.		4 10 1 1	Good. Ind.	0	0	Vomiting, pain in back, head- ache, las- situde.	r.	L. whole	Sov. 7 d. Sud.	R.	0	

L.R.C.P.	F.	Suft.	Bron. Cat. Erysip.	5 3	Ind.	F. d. bronch.	Measles 4 y. ago.	sore throat.	R. base.	7 d. Grad.	R.	Pleurisy.
130 E. Cureton, L.R.C.P.	23	Temp. Suft.	0		Good. Good.			Fever, pain in side.	0	Sev. 7 d.	10 d.	
131 C. Jordison, L.R.C.P.	50	Intemp. Suft.	—		Ind. Ind.	0	0	Erysip. face and L. chest.	r. L. base.	Sev. till D.	D.	
132 J. W. Lane, M.D.	22	Temp. Suft.	Pneum. Scarlat.	2 2	Bad. Bad.	0	0	Slight cough.	r. Both bases.	Mod. 7 d. Sud.	10 d.	0
133 W. H. Packer, L.R.C.P.	40	Temp. Suft.	Tonsill. Bron. Cat. In asylum of 520.	3 11	Good. Good.	—	" Abscess burst into lung over 12 y. ago."	Catarrh somo v.	0 Both apices.	Sev. till D.	D. 13th d	6
134 A. C. Malley, M.B.	40	Temp. Suft.	Tonsill. Bron. Cat.	1	Ind. Ind.	M. } bronch. 2 S. } Niced. phth.	—	Malaise.	r. L. base.	Sev. 5 d. Sud.	?	0
135 E. S. Scott, M.B.	2½	Tot. abs Suft.	Pneum. Enteric. Diphth. Tonsill. Bron. Cat.	2 + + 1 5	Good. Ind.	M.'s S. d. pneum.	0	Fever, drowsiness.	r. L. apex.	Sev. 7 d. Sud.	13 d.	Pleurisy.
136 "	25	Temp. Suft.	Pneum. Enteric. Tonsill. Bron. Cat.	2 + 1 4	Ind. Good.	0	Similar attack 3 y. ago.	Vomiting, stupor.	r. Both bases.	Sev. 7 d. Grad.	18 d.	—
137 M. Thomson, L.K.Q.C.P.	40	Temp. Suft.	Bron. Cat.	1	Good. Good.	1 B. d. phth.	Bronch. 6 y. ago.	0	r. R. base.	Sev. 6 d. Grad.	16 d.	0
138 G. B. Collett, L.R.C.P.	15	Temp. Suft.	Pneum. Measles. Catarrh. Tonsill. Herpes.	2 20 2 1 2	Ind. Ind.	0	Measles 3 y. ago.	Faintness, headache, shiverings.	r. L. base.	Mod. 6 d. Grad.	9 d.	—
139 "	5	Tot. abs Suft.	—		Ind. Ind.	F., 2 Bs., U., d. phth.	Bron. 2 y. ago, tonsill 1 m. ago.	Headache, sickness, pain in side.	r. L. base.	Mod. 6 d. Grad.	10 d.	—

1. His son had pneum. 2 w. before in same house. 2. 125 is daughter of 124 and was seized 3 d. after him. 3. Miscarriage (3rd m.) with much hemorrhage during crisis; attack ascribed to chill. 4. Peculiar case; very mild fever; two-thirds of both lungs affected; almost identical attack last year. 5. At first resembled scarlat. and had a rash. 6. P. M. Hepatization both apices; edema of bases; a lumatic; pneum. developed from a catarrh without rigors. 7. T. fell by crisis on 6th d. rose again to 102 on 7th with involvement of L. apex; stationary 15 d. then fell to 101; stationary 7 d.



Observer's Name.	Sex.	Age.	Temperate or other-wise. Food.	Concurrent cases of illness in	Same house.	Same district.	Sanitary state of house " " of district	Family history of lung disease.	Previous illnesses of patient.	Premontory symptoms.	Mode of onset.	Part of lungs affected.	Character of fever. { Duration of physical signs.	Result.	Sequelae.	Note.
140 G. B. Collett, L.R.C.P.	M.	42	Intemp. Suff.	—			Good. Good.	0	Gout 1 y. ago, and often before.	Sickness, malaise.	r.	L. base.	Sev. 21 d. Grad.	R.	—	1
141 R. L. Stokes L.R.C.P.	M.	38	Temp. Suff.	Pneum. Bron. Cat.	1 2		Ind. Ind.	0	Pneum. 16 y. ago.	Chilliness.	r.	L. base.	Sev. 7 d. Sud.	D. 9th d.		2
142 W. A. Hollis, M.D.	F.	40	Temp. Suff.	Enteric.	+		Good. Good.	0	0	Dyspnœa vomiting.	r.	Both wholes.	Sev. 5 d. Sud.	D. 7th d.		3
143 "	F.	33	Temp.? Suff.	Enteric. Measles.	1	+	? Good.	0	Delicate since measles 7 y. ago.	Sense of suffocation, vomiting, pain over heart and in joints.	r.	R. apex. L. whole	Sev. 4 d. Sud.	R.	Some slight signs at R. ap. on 30th d.	
144 H. G. Thompson, M.D.	M.	24	Temp. Suff.	Ac. Rheum. Pneum. Tonsill. Bron. Cat.	2 3 1 2		Ind. Good.	1 B. } d. phth. 1 S. }	Catarrhs.	0	r.	R. base.	Sov. 8 d. Sud.	R.	0	
145 "	M.	26	Temp. Suff.	Ac. Rheum. Enteric. Diphth. Catarrh. Tonsill. Bron. Cat. Erysip.	2 3 1 1 2 2 3 1		Bad. Good.	1 B. d. phth.	Catarrhs.	0	r.	L. base.	Sev. 8 d. Sud.	R.	0	
146 C. H. Watts Parkinson, M.R.C.S.	F.	62	Temp. Suff.	Pneum. Catarrh. Tonsill. Bron. Cat. Erysip.	1	+	Good. Good.	1 son double pneum.	Chron. cough. 4 times last few y., once badly both sides 2 y. ago.	Cough, cold, diarrhoea, fever.	r.	R. whole	Mild. 10 d. Gr. d.	R. Chr. pneum. part. phthisis.		



Observer's Name.	Sex.	Age.	Temperature or other- wise. Food.	Concurrent cases of illness in	Same house.	Same district.	Sanitary state of house " " of district	Family history of lung disease.	Previous illnesses of patient.	Prenatal history symptoms.	Mode of onset.	Part of lungs affected.	Character Duration of fever.	Duration of physical signs.	Result.	Sequelae.	Note.
159 E. T. Wilson, M.D.	M.	15	Temp. Suff.	Measles.		+	Good. Good.	M. tendency to phth.	0	Chilliness.	r.	L. base.	Sev. 5 d. Grad.	12 d.	R.	0	
160 W. L. Hunter, M.D.	F.	50	Temp. Suff.	Enteric. Scarlat. Bron. Cat.	2 1 5	2 1 5	Ind. Ind.	?	Ac. Rheum.	Catarrh.	r.	R. base.	Mod. 7 d. Sud.	-	R.	0	1
161 R. W. Mac- donald, M.B.	M.	42	Intemp. Suff.	Catarrh. Bron. Cat.	2 1	2 1	Good. Good.	?	Similar attack 20 y. ago, complained of L. lung ever since.	Headache pain L. lung, B. costive, anorexia.	r.	L. base	Sev. till D.	till D.	D. 5th d.		
162 "	M.	8	Temp. Suff.	Pneum. Catarrh. Bron. Cat	2 6 12	2 6 12	Good. Ind.	M. d. phth. F. bronch.	Pneum. 2 y. ago.	Pain head and back, B. costive.	0	R. whole L. base.	Mod. till D.	till D.	D. 5th d.		2
163 G. W. Joseph, M.R.C.S.	M.	21	Temp. Suff.	0			Good. Ind.	0	0	0	r.	Both bases.	Sev. till D.	till D.	D. 8th d.		3
164 J. F. Joseph, M.R.C.S.	M.	23	Intemp. Suff.	0			Bad. Bad.	M. d. bronch.	0	0	r.	R. base.	Sev. 8 d. Grad.	11 d.	R.	-	4
165 F. T. Thistle, L.R.C.P.	M.	28	Intemp. Suff.	Pneum.	1	1	Ind. Ind.	0	0	Headache, Malaise.	r.	L. base.	Sev. 10 d. Grad.	16 d.	R.	0	5
166 W. J. le Grand, M.D., Surg. A.M.D.	M.	22	Temp. Suff.	-			Good. Ind.	1 S. d. phth. F. bronch. twice.	Pneum. L. 2 y. ago.	Catarrh.	r.	L. base.	Sev. 9 d. Grad.	over 24 d.	R.	-	
167 A. do W. Baker, L.R.C.P.	M.	29	Temp. Suff.	Pneum.	4	4	Ind. Good.	M. d. bronch	0	Chilliness, malaise, pain in side.	r.	R. mid.	Mod. (?) Sud.	?	R.	-	
168 O. Bowen M.R.C.S.	F.	6½	Temp. Suff.	Varicella. Bron. Cat.	3 1	3 1	Good. Good.	F.'s family bronch.	Scarlat. } 3 y. Chorea } ago, measles } 2 y.	Vomiting.	0	R. base.	Sev. 9 d. Grad.	8 d.	R.	0	6



							her family.						to apex.			
170 A. B. Brabazon, M.D.	M.	22	Temp. Suff.	0			Good. Good.	0	Enterle, 6 m. ago, pulm. congestion.	Malaise.	r.	L. base.	r.	5 d.	R.	0
171 W. Vawdrey Lush M.D.	M.	38	Temp. abs. Suff.	Erysip.		1	Good. Good.	M. d. phth.	0	Cough.	0	L. base.	0	7 d.	R.	0
172 W. H. Williams M.D.	F.	43	Temp. Suff.	Catarrh.		3	Good. Good.	F. ehr. bronch M. d. phth.	Spasmodic asthma for years, nasal peristittis 1 m. ago.	General pains, stitch.	r.	L. whole	r.	—	D. 22nd d	8
173 P. Caldwell. Smith, M.B.	M.	49	Temp. Insuff.	Scarlat. Tonsill. Bron. Cat.	1	8 2 3	Bad. Bad.	0	<i>Double pneum.</i> 14 y. ago.	Tonsill. 2 d.	r.	Both bases.	r.	—	R. part.	9 Chronic pneum, L. lung.
174 W. J. Spence, L.R.C.P.	M.	3	Temp. Suff.	Pneum. in hosp.	3		Good. Good.	0	Bronch. 3 m. ago.	Restlessness, anorexia, fever.	0	R. base.	0	9 d.	R.	10 0
175 T. Bates, M.R.C.S.	F.	21	Temp. Suff.	Diphth. Scarlat. Enteric. Tonsill. Herpes. Bron. Cat. Erysip.	1 3 3 1 1 2 1		Good. Good.	0	Delicate. Amenorrhœa.	Headache.	0	Both wholes.	0	till D. 10th d	D.	
176 E. Thomson, M.B.(Omagh).	M.	22	Temp. Suff.	0			Ind. Ind.	0	0	0	r.	R. base.	r.	7 d.	R.	0
177 G. Chapman, M.R.C.S.	M.	34	Temp. Suff.	Tonsill. Bron. Cat.	+	+	Ind. Good.	—	0	—	r.	R. base.	r.	6 d.	R.	0
178 M. Moore, M.D.	F.	50	Temp. Suff.	Measles. Scarlat. Tonsill.	1 1 2		Good. Good.	—	Bronch. 1 y. ago, occas. rheum., am- putat. breast.	Fever.	0	R. base.	0	42 d.	R.	—

1. Nursing a fatal case of pneum. in another house; several adults died lately in same house. 2. Onset very sud.; fatal from first; no exp. to cold or damp, who nursed him, d. pneum. 8th day, Dec. 24th, in patient's house; aunt's child took pneum. L. base, Dec. 24; patient, who was often in aunt's room, and not exp. to cold or wet, took pneum. Dec. 25. 3. D. with typhoid symps. 4. Laryngitis also. 5. Drinking hard just before; plethoric. 6. No exposure; indoors 3 w. previous. 7. Uncle d. pneum. Dec. 16; aunt. 8. Rusty sput. ceased on 9th d., on 14th d. rigors and severe cerebral symps., lasting till D. 9. Great depression from wife's D. 1 m. before. 10. Prob. from exposure. 11. Onset 11 d. after amputation of breast.

Observer's Name.	Sex.	Age.	Temperature or other viso. Food.	Concurrent cases of illness in	Same house.	Same district.	Sanitary state of house " " of district.	Family history of lung disease.	Previous illnesses of patient.	Prenatal symptoms.	Mode of onset.	Part of lungs affected.	Character Duration Termination of fever.	Duration of physical signs.	Result.	Sequelæ.	Note.
179 Hugh R. Ker, F.R.C.S.	M.	11	Tot. abs. Suff.	Catarrh. (in the school).	12		Ind. Bad.	M. asthma.	Measles 6 y. ago, tonsill 4 y. ago.	Catarrh.	0	L. base.	Sev. 7 d. Grad.	12 d.	R.	0	
180 J. Bellingham, M.R.C.S.	M.	28	Temp. Suff.	0			Good. Good.	F. chr. bron.	Variola slightly.	Malaise.	r.	L. base.	Sev. 8 d. Grad.	16 d.	R.	0	
181 J. G. Clendinning, L.R.C.S.I.	M.	45	Temp. Suff.	Tonsill. Herpes. Erysip. Variola.		2 1 1 +	Bad. Ind.	0	0	H. adache	r.	R. base.	Mod. till D.	till D. 8th d.	D.		1
182 R. Peart, M.D.	M.	8	Tot. abs. Suff.	Catarrh. Rheum. Scarlat. Tonsill. Herpes. Erysip.	2	1 1 3 3 2	Good. Good.	0	0	Vomiting & Delirium 2 d.	0	L. base.	Mod. 5 d. Sud.	8 d.	R.	—	2
183 C. E. Hoar, M.D.	M.	74	Temp. Suff.	Bron. Cat.	1		Good. Good.	0	Gouty; chr. bron. every winter.	Anorexia, Apathy, Sleeplessness.	0	R. whole	Mod. till D.	till D. 5th d.	D.		3
184 T. W. H. Garg- tong, M.R.C.S.	M.	21	Temp. Suff.	Pneum. Enteric. Bron. Cat.		1 1 3	Good. Good.	2 Bs. d. plth.	Pleurisy 3 w. ago.	0	r.	L. base.	Sev. ? Grad.	?	R.	R. pneum. Hydro tho- rax, and empyema.	4
185 "	F.	7 m.	Tot. abs. Suff.	Pneum. Scarlat. Rheum. Catarrh. Tonsill. Erysip.		2 1 3 3 1 10	Ind. Ind.	0	0	Malaise 2 d.	0	Both bases.	Mod. till D.	till D. 7th d.	D.		





Observer's Name.	Sex.	Age.	Temperate or other-wise. Food.	Concurrent cases of illness in	Same house.	Same district.	Sanitary state of house " " of district	Family history of lung disease.	Previous illnesses of patient.	Premontory symptoms.	Mode of onset.	Part of lungs affected.	Character of fever. { Duration Termination	Duration of physical signs.	Result.	Sequelæ.	Note.
195 W. Fred. Dix, M.R.C.S.	M.	23	Temp. Suff.	Pneum. Catarrh.	4		Ind. Ind.	—	—	Catarrh.	0	Both bases.	Sev. 12 d. Grad.	15 d.	R.	—	1
196 "	F.	20	Temp. Suff.	" "	"		" "	—	Tonsill 1-y. ago.	Catarrh.	0	L. apex.	Sev. 12 d. Grad.	14 d.	R.	—	
197 "	F.	18	Temp. Suff.	" "	"		" "	—	—	Catarrh.	0	L. base	Mod. 9 d. Grad.	11 d.	R.	—	
198 " P. Eade, M.D. }	M.	52	Temp. Suff.	" "	"		" "	—	—	Catarrh.		R. base. L. whole	Mod. 6 d. Grad.	9 d.	R.	—	
199 J. Munro, M.D.	M.	29	Temp. Suff.	Pneum. Tonsill. Febricula. Catarrh.	1	3	Bad. Bad.	0	0	0	r.	R. whole L. base.	Sev. 8 d. Grad.	15 d.	R.	0	3
200 "	M.	19	Intemp. Suff.	Pneum. Febricula. Tonsill.	1	3	Good. Good.	M. weak chest. B. chr. bron.	0	0	r.	Both bases.	Sev. 10 d. Grad.	42 d.	R. part.	R. base only partially cleared, will prob. soften.	
201 "	F.	14	Tot. abs. Suff.	Pneum. Febricula. Catarrh. Tonsill. Bron. Cat.		3	Good. Good.	0	Scarlat. 3 y. ago, rubeola 5 y. ago.	Febricula 5 d.	r.	L. whole	Sev. — Grad.	over 90 d.	R.	0	4
202	M.	56	Temp. Suff.	Scarlat. Rötheln. Bron. Cat. Erysip.	2	+	Good. Good.	—	Occas. bron.	Hent, head-ache, nausea.	r.	Both bases.	Mod. 7 d. Grad.	28 d.	R.	0	
203 J. W. Lane, M.D. }	M.	72	Temp. Suff.	Pneum.		1	Bad. Bad.	—	—	Pain, cough, dyspnea.	r.	Both bases.	Sev. till D.	ti'l D.	D.	—	5
204 "	F.	53	Temp.	Pneum.	2		Bad.	—	Ac. rheum.	Cough, pain.	r.	Both	Sev.	till D.	D.	—	



Observer's Name.	Sex.	Age.	Temperato or other- wise. Food.	Concurrent cases of illness in	Same house.	Same district.	Sanitary state of house " " of district	Family history of lung disease.	Previous illnesses of patient.	Premontory symptoms.	Mode of onset.	Part of lungs affected.	Character of fever Termination	Duration of physical signs.	Result.	Sequelæ.	Notes.
216 E. Skinner, L.R.C.P.	F.	8	Tot. abs. Suff.	—			Bad. Good.	F. chr. bron.	0	0	r.	L. base.	Mild. 3 d. Grad.	10 d.	R.	0	
217 W. E. Lascombe, L.R.C.P.	M.	6	Temp. Suff.	Tonsill. Bron. Cat. Erysip.	1 ++ 3		Good. Good.	0	Scarlat. 3 y. ago.	Languor, anorexia.	r.	R. apex.	Mod. 8 d. Grad.	14 d.	R.	0	
218 C. P. Coombs, M.D.	M.	8	Tot. abs. Suff.	Catarrh. Tonsill. Bron. Cat.	1 3 ++		Good. Good.	0	Febricula.	—	r.	R. base.	Sev. 8 d. Sud.	over 7 d.	R.	0	
219 R. J. H. Scott, M.R.C.S.	M.	11	Temp. Suff.	Tonsill. Bron. Cat.	1 1		Good. Good.	M's. fam. phthisical.	Debility, scarlat., measles.	Cough, tightness in chest.	r.	R. base.	Mod. 8 d. Sud.	7 d.?	R.	Tonsill.	
220 A. H. Bampton, M.D.	F.	32	Temp. Insuff.	Bron. Cat.	3		Ind. Ind.	0	Bronch. often, crysip. 4 m. ago.	Anorexia, pain L. breast and head.	r.	Both bases.	Sev. 7 d. Grad.	14 d.	R.	0	1
221 R. L. Batten- bury, M.D.	M.	17	Temp. Suff.	Catarrh. Tonsill. Bron. Cat.	1 2		Good. Ind.	M. and M's. M. phth.	Varicella } 5 y. measles } ago hæmopt. } y. ago.	Malaise 2 w.	r.	R. base.	Mod. 7 d. Sud.	12 d.	R.	—	
222 G. A. Cardew, M.R.C.S.	F.	46	Temp. Suff.	Measles. Bron. Cat.	1		Good. Ind.	—	Bronch. often, ac. nephrit. 6 w. ago.	Pain in side.	r.	L. base.	Mod. 7 d. Sud.	17 d.	R.	—	2
223 "	F.	7 m.	Tot. abs. Suff.	Pertuss. Bron. Cat.	3 1		Good. Good.	2 older ch. bron.	Pertuss. for 2 m. before.	Restless- ness, pain.	0	R. base.	Mod. 11 d. Sud.	28 d.	R.	Bronchitis 3 w. later.	
224 A. A. Cohen, M.D.	F.	7	Temp. Suff.	Pneum. Tonsill. Bron. Cat.	3 4 6		Ind. Ind.	M's. U. d. phth. at 23. F's. M. d. phth. at 25. S. acuto	Phth. 2 y.	Vomiting.	r.	R. base.	Sev. 8 d. Sud.	11 d.	R.	0	



Case	Complainant	Sex	Age	Temp.	Pulse	Respir.	General	3 A. d. phth.	Wet lung.	Cough, &c.	r.	R. base.	Sud.	13 d.	R.	0	3
226	F.R.C.S.	F.	49	Temp. Suff.	"	"	"	M. Bron.	0	Cold 14 d.	r.	R. base.	Sev. 6 d. Sud.	13 d.	R.	0	3
227	T. Corbett, M.R.C.S.	M.	47	Temp. Suff.	1	Bron. Cat.	"	0	0	Catarrh 4 d.	r.	R. base.	Sov. 4 d. Grad.	12 d.	R.	—	
228	D. A. Davies, M.B.	M.	19	Tot. abs. Suff.	"	0	Ind. —	0	Fever 3 y. ago.	Headache 1 d.	r.	R. apex.	Sev. 12 d. Grad.	over 20 d.	R.	—	4
229	E. Drummond, M.D.	F.	30	Temp. Suff.	"	—	Ind. Good.	F. d. phth.	Delicate.	Languor, sleeplessness, anorexia.	r.	L. base.	Sov. 7 d. Grad.	12 d.	R.	0	
230	A. P. Fiddian, M.B.	M.	7	Temp. Suff.	1 2	Tonsill. Bron. Cat.	Ind. Ind.	The baby has bron.	Measles, { 6 y. ago. Pertussis, } ago.	Vomiting.	—	Both bases.	Mild. 6 d. Grad.	8 d.	R.	0	5
231	C. Firth, M.D.	M.	12	Temp. Suff.	1 +	Pneum. Rthel. Bron. Cat.	Ind. Ind.	—	—	Sickness, headache, pain in chest.	0	Both bases.	Sev. 7 d. Sud.	8 d.	R.	—	6
232	J. W. Gooch, M.R.C.S.	M.	16	Temp. Suff.	5 16	Pneum. Scarlat. Catarrh. Tonsill. Bron. Cat. Herpes.	Good. Good.	0	0	Cough, shivering, pain in side.	r.	R. base.	Mod. 5 d. Sud.	14 d.	R.	0	
233	A. Halloves, M.R.C.S.	M.	29	Temp. Suff.	1	—	Good. Good.	0	Pneum. 6 y. ago.	0	r.	L. base.	Mod. 9 d. Sud.	11 d.	R.	0	
234	J. Hamilton, M.D.	M.	18	Temp. Suff.	1 3 1 1 2 4 2 10	Pneum. Scarlat. Diphth. Variola. Roseola. Catarrh. Herpes. Bron. Cat.	Ind. Ind.	F's. M. d. phth. at 24.	0	0	r.	Both bases.	Sev. till D. 11th d.	till D. 11th d.	D.	7	

1. Got wet washing. 2. Got wet washing. 3. Cases 225, 226, were in the same house at the same time. Another case of pneum. next door. 4. Onset on board ship. 5. Out in a procession on a cold day. 6. Onset sudden with no apparent cause. 7. Coma on 2nd d.; conscious on 4th d.; coma again on 5th d. and D. in that condition.

Observer's Name.	Sex.	Age.	Temperate or otherwise. Food.	Concurrent cases of illness in	Same house.	Same district.	Sanitary state of house " " of district	Family history of lung disease.	Previous illnesses of patient	Premontory symptoms.	Mode of onset.	Part of lungs affected.	Character of fever. Duration of fever.	Duration of physical signs.	Result.	Sequelae.	Note.
235 E. W. Hope, M.D.	F.	6	Tot. abs. Suff.	Bron. Cat.	1		Ind. Ind.	F. } coughs. M.	Bronch. oft.	Pain in side, anorexia, restlessness.	r.	L. base.	Sev. 6 d. Sud.	12 d.	R.	0	
236 Hugh R. Ker, F.R.C.S.	M.	14	Tot. abs. Suff.	0			Ind. Ind.	F. bron. B. d. bronchopneum at 2.	Bronch. oft.	Pain in head, cough, fever.	r.	R. base.	Mild. 6 d. Grad.	over 9 d.	R.	—	
237 K. N. McDonald, M.D.	F.	65	Temp. Suff.	Catarrh. Tonsill. Bron. Cat.	2 2 1		Good. Good.	F. d. pneum. at 7 l.	None for 30 y., convalesce. fr. fract. R. tibia.	0	r.	Both bases.	Sev. 11 d. Grad.	14 d.	R.	0	
238 Donald McLeod, M.D.	M.	56	Temp. Suff.	0			Ind. Good.	0	Sciatica 6 y. ago.	Pains, chills, anorexia, thirst.	r.	R. apex. L. base.	Mod. 8 d. Grad.	14 d.	R.	0	
239 J. M. H. Martin, M.D.	M.	70	Temp. Suff.	Fever. Bron. Cat.	1 1		Good. Ind.	0	Cardiac dropsy.	Chills, headache, muscular pains.	r.	L. base.	Mod. 16 d. Grad.	18 d.	R.	—	
240 Robert Mears, L.R.C.P.E.	F.	—	Tot. abs. Suff.	Pneum. Tonsill. Bron. Cat.	1 1 3		Ind. Good.	B. d. ae. pneum. 3 d. before onset of pat.'s illness.	Variola 5 y. ago.	Pain in side, chill, malaise.	r.	Both bases.	Sev. till D.	till D. 10thd.	D.		1
241 A. Napier, M.D.	F.	25	Temp. Suff.	0			Good. Ind.	0	0	Pain in side.	r.	R. base.	Mod. 7 d. Sud.	2 d.	R.	0	
242 J. B. Neal, M.R.C.P.E.	M.	31	Temp. Insuff.	Pneum.	1		— —	0	0	0	r.	R. whole	Mod. 9 d. Sud.	34 d.	R.	Syst. mitr. bruit. Not heard before.	

		Surf.			Good.	1 B. subac. pneum. 2 y. ago.	cere, crysip. 8 y. ago, pleurisy 2 y. ago.		r.	R. whole	10 d. Grad.	
245 G. E. Powor, M.R.C.S.	M.	Tot. abs. Suff.	12	Tonsill. Bron. Cat.	+	+	0	—	0	R.	Sev. 8 d. Sud.	—
246 J. E. Ranking, M.D.	F.	Tot. abs. Suff.	5½	0	Good. Good.	0	0	Langnor, vomiting, anorexia, fever.	0	L. base.	Mod. 6 d. Sud.	0
247 W. Sellers, jun., M.B.	M.	Temp. Suff.	55	Tonsill. Bron.	+	+	—	Malaise, cough, dys- pnoea.	r.	R. base.	Sev. 10 d. Sud.	0
248 H. T. Sells, M.R.C.S.	M.	Tot. abs. Suff.	32	—	Good. Good.	0	0	—	r.	Both bases.	Sev. till D.	D. 10thd.
249 A. Shoen, M.D.	M.	? Suff.	34	Bron. Cat.	1	Good. Good.	0	Aching, head- ache, pain in side.	r.	R. base.	Mod. 3 d. Sud.	18 d.
250 R. Smith, F.R.C.S.E.	F.	Temp. Suff.	69	Tonsill. Bron. Cat. Ac. Rheum.	1 6	Bad. Ind.	—	Langnor, stiffness, bruised feeling.	r.	R. base.	Mod. 4 d. Grad.	9 d.
251 S. C. Smith, M.D.	F.	Temp. Suff.	20	0	Good. Ind.	0	Spasm. asthma 4 y. ago.	Cold & cough 3 w., pain in side.	0	Both bases.	Sev. 7 d. Sud.	36 d.
252 W. H. R. Stanley, M.D.	F.	Temp. Insuff.	50	Scarlat. Catarrh. Tonsill. Bron. Cat.	3 6 3 2	Ind. Good.	0	—	r.	L. base.	Sev. 14 d. Grad.	14 d.
253 T. W. Thursfield, M.D.	F.	Temp. Suff.	37	Pneum. Enteric. Scarlat. Diphth. Erysip.	2 1 1 1 1	Ind. Good.	0	Catarrh.	r.	R. apex.	Sev. 8 d. Sud.	20 d.
												0
												Pericarditis.

1. Pat.'s B., a healthy man previously, d. of pneum. at a village 5 miles off; Pat. visited him 3 d. before onset of her illness. 2. Pat., a lunatic in Asylum. Pneum. occurred during attack of acute delirious mania with refusal of food, and insomnia. D. from rapid inanition, insomnia, and excitement. 3. Exp. in a snowstorm night before onset.



Observer's Name.	Sex.	Age.	Temperate or otherwise. Food.	Concurrent cases of illness in	Same house.	Same district.	Sanitary state of house " " of district	Family history of lung disease.	Previous illnesses of patient.	Fromontory symptoms.	Mode of onset.	Part of lungs affected.	Character of fever. { Duration Termination } of fever.	Duration of physical signs.	Result.	Sequelæ.	Note.
254 Th. W. Thursfield, M.D.	M.	8	Tot. abs. Suff.	Pneum. Enteric. } Scarlat. Tonsill. Herpes. Bron. Cat.		2 3 1 1 1	Good. Good. Good.	G. M. d. pneum. S. d. ac. hydropneum. at 1 y.	Scarlat. 7 m. ago.	Catarrh.	r.	L. base.	Mod. 6 d. Sud.	14 d.	R.	0	
255 J. W. Wolfenden, L.R.C.P.	M.	—	Temp. Suff.	Pneum. Fevcr. Tonsill. Bron. Cat. Erysip.	4	2 4 1 1	Ind. Good.	0	0	Dyspepsia.	r.	R. whole L. base.	Sev. 18 d. Grad.	16 d.	R.	—	1
256 T. L. Gentles, L.F.P.S.	F.	37	Temp. Suff.	Scarlat. Erysip.		+ 3	Good. Good.	1 S. Intemp. d. phth.	0	Hoarseness, vomiting.	r.	L. base.	Sev. 7 d. Sud.	16 d.	R.	Debility.	
257 E. T. Tylocoto, M.D.	M.	56	Temp. Suff.	—			Good.	0	0	0	r.	R. base.	Mod. 12 d. Grad.	16 d.	R.	0	
258 T. G. Lithgow, L.R.C.P.	M.	65	Temp. Suff.	Pneum. Erysip.		1 1	Ind. Good.	—	Bronch. and rheumat. yearly.	—	r.	Both bases.	Mod. till D. till D.	till D.	D. 7th d.		
259 Duncan J. Mackenzie, M.D.	F.	11	Temp. Insuff. (?)	Pleurisy.	1		Bad. Bad.	M. d. prob. of pneum. "ill S. d. with cough."	Measles 1 y. ago.	Headache, dizziness, pain in side.	0	R. base.	Sev. 7 d. Grad.	10 d.	R.	0	
260 Stanley Taylor, M.B.	M.	31	Temp. Suff.	—			Ind. Ind.	1 S. d. "decimo."	0	Catarrh.	0	L. base.	Sev. 12 d. Grad.	21 d.	R.	—	2
261 J. Aikman, M.D.	F.	26	Tot. abs. Suff.	Scarlat. Enteric. } Pertussis		5 +	Ind. Ind.	F. phth. 1 B. 11 phth. 1 S. 17 phth. 1 S. 6 m. marasmus.	Dyspep. and jaundice 4 y. ago.	Cough, pain in side.	r.	L. apex.	Sev. 8 d. Sud.	20 d.	R.	0	
262 J. F. Allen, M.D.	M.	54	Intemp.	Pneum.		2	Good.	0	0	0	r.	R. whole	Sev.	till D.	D.		3

264 A. H. Hampton, M.D.	F.	40	Temp. Suff.	Bron. Cat.	1	Ind. Bad.	0	0	Chill.	r.	R. base.	Grad. Mod. 8 d. Grad.	16 d.	R.	0	4
265 W. R. Bates, L.K.Q.C.P.	F.	58	Temp. Suff.	Pneum.	3	Ind. Ind.	1 S. d. phth. 40. 1 B. d. phth. 28.	Chr. bronch. and emphy- sema.	0	r.	Both bases.	Mod. 8 d. Sud.	—	R.	0	
266 R. L. Batterbury, M.D.	F.	66	Temp. Suff.	Pneum.	3	Good. Good.	—	Bronch. 3 y. ago, chronic rheum.	0	r.	R. base.	Sev. 6 d. Grad.	till D.	D. sth d.		
267 "	M.	35	Temp. Suff.	Pneum.	3	Good. Good.	F. d. pleur.	Pneum. 23 y. ago, pleuro- pneum. 4 y. ago.	Nausea.	r.	R. base.	Mod. 6 d. Grad.	10 d.	R.	—	
268 "	M.	20	Temp. Suff.	Pneum.	3	Good. Good.	0	0	0	r.	L. base.	Sev. 6 d. Sud.	10 d.	R.	—	
269 M. G. Biggs, M.R.C.S.	M.	42	Tot. abs. (prev. in- temp. Suff.)	Pertussis. Broncho- pneum. Scarlat. Measles. Tonsill. Bron. Cat.	6 1 1 3 22	Ind. Ind.	F. chr. bron.	Chr. bronch.	Cough, pain in side.	r.	L. base.	Mod. —	—	D. sth d.		5
270 G. Black, M.B.	M.	24	Temp. Suff.	0		Good. Good.	F. chr. bron.	0	Vomiting, lumbar pain.	0	L. apex.	Sev. 12 d. Grad.	6 d.	R.	0	
271 "	M.	59	Temp. Suff.	Pneum. Erysip.	2 1	Good. Good.	—	Chr. rheum.	Giddiness, diarrhea for 8 h.	r.	R. base.	Mod. till D.	till D.	D. 7th d.		6
272 J. Mackenzie Booth, M.D.	M.	21	Tot. abs. Suff.	0		Good. Ind.	F's. fam. phth.	Inflamm. of lung, once.	Headache, delirium, epistaxis.	r.	Both bases.	Sev. 11 d. Sud.	20 d.	R.	0	
273 "	M.	21	Tot. abs. Suff.	—		Bad. Bad.	0	0	Chills, rest- lessness, vomiting, pain.	r.	L. base.	Sev. 4 d. Sud.	8 d.	R.	0	

1. Congestion of liver and jaundice 4th d. 2. Wet through night before onset. Wife had pneum. 14 d. before. 3. D. by collapse. Diarrhea, sweating, brown tongue, of typhoid character. 4. Exp. to wet. 5. D. by asphyxia. 6. Mental depression owing to losses and D. of wife shortly before. 7. Epistax. profuse and repeated 1 d. before and 5 d. aft. onset.

Observer's Name.	Sex.	Age.	Temperate or otherwise. Food.	Concurrent cases of illness in	Same house.	Same district.	Sanitary state of house " " of district	Family history of lung disease.	Previous illnesses of patient.	Premontory symptoms.	Mode of onset.	Part of lungs affected.	Character of fever. (Duration termination)	Duration of physical signs.	Result.	Sequelæ.	Note.
274 J. Mackenzie Booth, M.D.	F.	6	Tot. abs. Suff.	—			Good. Ind.	M's M. d. phth.	Pertuss. } 4 y. Measles. } ago. Bron.	Headache, chilliness.	r.	R. base.	Sev. 3 d. Sud.	11 d.	R.	0	
275 "	M.	7	Tot. abs. Suff.	Bron. Cat. Measles.		++	Bad. Bad.	0	0	0	r.	R. base.	Sev. 10 d. Sud.	14 d.	R.	0	
276 J. G. Clendinnen L.R.C.S.	M.	25	Intemp. Suff.	Variola.		+	Good. Ind.	F. d. bron.	0	Headache, tremors, sickness.	r.	R. base L. whole	Sev. 8 d. Grad.	12 d.	R.	0	
277 "	M.	29	Temp. Suff.	Variola.		+	Good. Ind.	M. d. bron.	0	Headache.	r.	R. whole	Sev. 7 d. Sud.	—	D. 10th d		1
278 "	M.	57	Temp. Suff.	Variola.		+	Good. Ind.	—	Bron. } yearly Asth.	Malaise, headache.	0	L. base.	Mild 2 d. Grad.	4 d.	R.	0	
279 A. A. Cohen, M.B.	M.	2	Tot. abs. Suff.	?			Ind. Ind.	M. pneum. 13. r's F. bron. M's F. d. phth. 56.	Pertuss 1 y. ago.	Poorliness.	r.	R. base.	Sev. 8 d. Sud.	12 d.	R.	0	
280 D. W. Craiz, M.D.	F.	11	— Suff.	—			Good. Good.	0	0	Diarrhoea, vomiting.	r.	R. base.	Sev. till D.	till D. 7th d.	D.		
281 E. Crossman, L.R.C.P.	M.	81	Temp. Suff.	0			Good. Ind.	Bron. common in fam.	Congest. both lungs 4 y. ago	Flushing, dyspnoea.	r.	R. base. L. whole	Sev. 7 d. Grad.	14 d.	R.	0	
282 A. and T. Davidson, M.B., M.B.	M.	—	Temp. —	Tonsill. Catarrh. Bron. Cat.	3	1 2 8	Good. Good.	M. bron. 1 B. repeated slight pneum.	0	Cough 1 m.	r.	R. base.	Mod. 11 d. Grad.	28 d.	R.	0	
283 D. Goyder, M.D.	M.	41	Temp. Suff.	Pneum. Catarrh. Tonsill.		2 1 1	Good. Good.	0	Typhus 25 y. ago, pneum. (slight) 8 y. ago	"Starved," pains, anorexia.	r.	R. base. L. whole	Sev. 4 d. Sud.	14 d.	R.	0	2
284 W. E. Green, M.D.	M.	27	Temp. Suff.	Measles.		+	Good.	B. phth.	0	Catarrh 3 w.	r.	R. base.	Sev.	20 d.	R.	0	



286	"	F.	7	— Suff.	0		Good. Good.	0	0	Sickness.	r.	R. base.	Grad. Mod. 3 d. Sud.	9 d.	R.	0
287	"	F.	48	Temp. Suff.	Measles.	+	Good. Good.	0	0	Congest. of lungs 4 y. ago.	?	Both bases.	Sev. 9 d. Grad.	18 d.	R.	0
288	C. Harrison, M.D.	M.	45	Intemp. Suff.	Pneum. Scarlat. Erysip.	1 +	Ind. Good.	0	0	Winter cough.	r.	Whole of both.	Sev. till D. till D.	D. 6th d.		4
289	"	M.	14	Tot. abs. Suff.	Pneum. Scarlat. Erysip. Bron. Cat.	1 + 2 +	Good. Good.	F. catarrhs, 1 ch. d. croup.	Bron. oft.	Cold feet, chills, shivering, pain in back.	r.	L. whole	Sev. 9 d. Grad.	11 d.	R.	0
290	H. W. Hubbard, L.R.C.P.	M.	56	Temp. Suff.	Enteric.	+	Good. Bad.	0	0	Erysip. in foot.	r.	R. base L. whole	Sev. 6 d. Sud.	14 d.	R. part.	Solidity of L. base.
291	"	M.	20	Temp. Suff.	0		Ind. Ind.	0	0	—	r.	Both bases.	Sev. 7 d. Sud.	10 d.	R.	0
292	C. J. B. John- son, L.R.C.P.	M.	39	Temp. Suff.	Pneum.	1	Good. Good.	0	0	Nothing for 20 y. save catarrhs for last 2 m.	r.	R. base.	Sev. 4 d. Grad.	14 d.	R.	0
293	F. J. Joynes, M.R.C.S.	F.	57	Temp. Suff.	Pneum.	4	Ind. Good.	2 B. d. phth. at 22.	0	0	r.	R. base.	Mod. 7 d. Sud.	17 d.	R.	0
294	W. J. Le Grand, M.D., Surg. A.M.D.	F.	24	Temp. Suff.	Pneum. Mumps. Bron. Cat. Erysip.	5 20 10 2	Go d. Ind.	0	0	Constipat. thirst, headache, pains.	r.	R. whole L. base.	Sev.	till D.	D.	
295	Th. McClure, M.D.	M.	5	Tot. abs. Suff.	Pneum. Tonsill. Bron. Cat.	3 1 +	Good. Good.	F. d. phth. M.'s fam. also phth.	Bron 2 y. ago.	Running of eyes, malaise.	0	Both bases.	Sev. 4 d. Sud.	9 d. (?)	R.	0

1. Prostration after sudden and violent palpitation. 2. 8 y. ago pneum. common among wool-sorters, which this man then was. 3. Exp. to cold; epileptic fits, averaging 600 per ann., always stop during illness. 4. Exp. to cold. 5. Working at drains the day before, then sick; physical signs cleared grad. in about 2 m.

Observer's Name.	Sex.	Age.	Temperature or other- wise. Food.	Concurrent cases of illness in	Same house.	Same district.	Sanitary state of house " " of district.	Family history of lung disease.	Previous illnesses of patient.	Prodromitory symptoms.	Mode of onset.	Part of lungs affected.	Character Duration Termination of fever.	Duration of physical signs.	Results.	Sequelae.	Notes.
296 James Neal, M.D. (Sam- down).	M.	8	Tot. abs. Suff.	Measles.	+	+	Good. Good.	0	Measles at the time.	0	r.	Both bases.	Sev. till D.	till D.	D. 17th d.		
297 W. J. Pilcher, F.R.C.S.	M.	41	Temp. Insuff.	Pneum. Erysip.	3 3	3 3	Bad. Good.	M. asthma.	—	Catarrh.	r.	Both bases.	Sev. 10 d. Grad.	12 d.	R.	0	
298 A. Ransome, M.D.	M.	14	Temp. Suff.	Catarrh. Tonsill.	+	+	Good. Ind.	U. d. phth.	Measles 2 y. ago.	Heaviness, headache.	r.	L. whole	Sev. 10 d. Grad.	—	R. part.	Empyema.	
299 C. Mason Scott, F.R.C.S.	F.	53	Temp. Suff.	Influenza. Bron. Cat.	+	5	? Good.	M. d. pneum. 1 S. chr. bron.	Nasal polypi, ailing some time.	0	r.	R. base, L. whole	Sev. till D.	till D.	D. 7th d.		
300 C. E. Shelly, M.B.	M.	42	Temp. Insuff.	Pneum. Catarrh. Tonsill. Herpes. Erysip. Bron. Cat.	4 5 15 1 2 12	4 5 15 1 2 12	Good. Ind.	—	Enteric 1 y. ago, poorly 4 w.	Vomiting, headache.	r.	Both bases.	Mod. till D.	till D.	D. 7th d.		
301 "	M.	23	Temp. Suff.	Pneum. Catarrh. Tonsill. Bron. Cat. Erysip.	1 5 6 13 9 3	5 6 13 9 3	Good. Ind.	0	0	Weakness 2 d., vomit- ing 12 h.	r.	L. base.	Mod. 6½ d. Sud.	9 d.	R.	Maniacal delir. with P. 47-60, and T. 96.5 for 2½ d. aft. crisis.	
302 "	M.	15	Temp. Suff.	Pneum. Catarrh. Tonsill. Bron. Cat. Erysip.	5 5 13 11 2	5 5 13 11 2	Good. Ind.	0	0	Headache.	r.	L. base.	Mod. 4 d. Sud.	10 d.	R.	0	
303	M.	16	Temp. Suff.	Pneum. Catarrh. Tonsill.	5 5 14	5 5 14	Good. Ind.	M. d. pneum. 1 B. d. phth. Nis. & T's. fam.	Pharyngitis 1 m. ago. difficite.	0	r.	Both bases.	Sev. till D.	till D.	D. 14th d.	—	





Observer's Name.	Sex.	Age.	Temperate or otherwise. Food.	Concurrent cases of illness in	Same house.	Same district.	Sanitary state of house " " of district	Family history of lung diseas.	Previous illnesses of patient.	Premontory symptoms.	Mode of onset.	Part of lungs affected.	Character of fever. { Duration Termination	Duration of physical signs.	Result.	Sequelae.	Note.
315 E. T. Wilson, M.B.	M.	17	Temp. Suff.	Pneum. Catarrh	1	3	Good. Good.	0	Enteric 10 y. ago, variola 8 y. ago.	Pain in side, epistaxis.	r.	R. base.	Sev. 7 d. Sud.	13 d.	R.	0	1
316 "	M.	12	Tot. abs. Suff.	Bron. Cat. Diarrhoea.	1	1	Ind. Ind.	G. F. } d. G. M. } phth. B. bron. oft.	L. ribs fract. 1 y. ago.	Pain in back.	0	L. base.	Mod 2 d. Sud.	20 d.	R.	0	
317 J. M. H. Martin, M.D.	M.	47	Temp. Suff.	Enteric.	+	+	Good. Good.	1 S. bron.	Chr. rheum.	Catarrh.	r.	R. base.	Mod. 6 d. Sud.	14 d.	R.	—	
318 "	F.	32	Temp. Suff.	Enteric.	1	1	Ind. Ind.	Many d. of pneum.	Bronch. 3 m. ago.	Catarrh.	r.	R. apex L. whole	Sev. —	—	D. 8th d.	—	2
319 W. Bernard, F.K.Q.C.P.	F.	26	Temp. Suff.	0			Good. Good.	F. d. phth.	Strumous.	0	0	R. base.	Mod. 10 d. Grad.	15 d.	R.	—	
320 J. Mackenzie Booth, M.D.	F.	58	Tot. abs. Insuff.	Pneum. Typhus. Measles. Tonsill. Bron. Cat.	1	2 3 + + few.	Ind. Ind.	F { chr. bron. and con- seq. morb. cordis.	Chr. bron., emphy., hypert. and dilat. R. heart.	Anorexia.	r.	R. apex.	Mod. till D. till D.	till D.	D. 4th d.	—	
321 "	M.	15	Tot. abs. Insuff.	Pneum. Typhus. Tonsill. Bron. Cat.	1	1 + + +	Ind. Ind.	M's F. and M's M. d. lung diseaso consequ. on morb. cordis.	Chr. bron., emphy.	Chilliness, delirium.	r.	Both bases.	Mod. 7 d. Grad.	21 d.	R.	—	
322 J. Bridger, M.R.C.S.	F.	18	Temp. Suff.	Pneum. Catarrh.		3 55	Good. Ind.	0	Influenza 1 m. ago.	0	r.	R. whole	Sev. 12 d. Grad.	16 d.	R.	0	
323 R. L. Batterbury, M.D.	F.	16	Tot. abs. Suff.	—			Good. Good.	0	0	Headache, sickness.	r.	R. base.	Mod. 7 d.	10 d.	R.	—	

325	"	F.	53	Temp. Suff.	Tonsill. Herpes. Bron. Cat. Erysip.	1	+	+	+	Good. Good.	0	Congest. liver, head-aches oft.	Chilliness.	0	R. whole	Sev. till d.	till D. 8th d.	D.	—	4	
326	S. Wellesley Coombs, F.R.C.S.E.	M.	45	Intemp. Insuff.	0					Bad. Bad.	1 B. d. pleur.	0	0	Catarrh 14 d.	r.	L. base.	Mod. 4 d. Sud.	9 d.	R.	—	
327	"	M.	32	Temp. Suff.	0					Bad. Bad.	0	Abscess in face 6 m. ago.	Cough 14 d.	r.	R. base.	Sev. 7 d. Sud.	14 d.	R.	R. otorrhœa swelling R. face.		
328	"	M.	23	Temp. Insuff.	0					Bad. Bad.	0	Pleuro-pneum. 5 y. ago.	Pain in side.	0	L. base.	Mod. 5 d.	10 d.	R.	—		
329	"	M.	13	Temp. Suff.	Enteric.	+				Bad. Ind.	0	Bronch. 1 y. ago.	Pains in back and legs.	r.	L. base.	Mod. 6 d. Sud.	10 d.	R.	0		
330	"	F.	10	Temp. Suff.	Pneum. Enteric.	1	+			" "	0	0	Languor, Fatigue.	0	R. apex.	Mod. 5 d. Grad.	10 d.	R.	0	5	
331	G. W. Crowe, M.D.	M.	22	Temp. Suff.	Enteric.	4				Ind. Ind.	0	"Congest. brain" 11 y. ago, subj. to vomiting and vertigo.	0	r.	Both bases.	Sev. 6 d. Grad.	16 d.	R.	0	6	
332	J. G. Douglas Kerr, M.B.	M.	66	Temp. Suff.	Pneum.	1				Good. Good.	0	Mental derangement, mitr. regurg. murmur.	Cold, cough, pain in side, fever, sleeplessness.	0	R. base.	Sev. till d.	till D. 7th d.	D.	—	7	
333	G. M. Edmond, M.D.	M.	37	Temp. Suff.	Pneum. Bron. Cat. Erysip.	6	3	2		Good. Good.	B. pneum. twice, 1 ch. pneum. once, 1 ch. sev. bronch.	0	Weakness, pain in back and legs.	r.	L. base.	Sev. 9 d. Grad.	16 d.	R.	Sev. myalgia of calf-muscles.		

1. Albuminur. 1/6 on 6th d.; none on 8th d. 2. Seemed fatal from onset. 3. Much albuminur.; granular casts; not exam. before attack. 4. Albuminur.; granular casts. 5. Sister to 329; her onset was 6 d. after his. 6. From chill; D. by collapse. 7. Ditto.

Observer's Name.	Sex.	Age.	Temperate or other- wisc. Food.	Concurrent cases of illness in	Same house.	Same district.	Sanitary state of house " " of district	Family history of lung disease.	Previous illnesses of patient.	Prodromitory symptoms.	Mode of onset.	Part of lungs affected.	Character Duration Termination of fever.	Duration of physical signs.	Result.	Sequelae.	Note.
334 T. F. Raven, L.R.C.P.	F.	27	Temp. Suff.	Enteric.		1	Good. Good.	0	0	Catarrh.	0	L. apex.	Mod. 6 d. Sud.	14 d.	R.	—	1
335 F. C. Fisher, M.R.C.S.	F.	21	Tot. abs. Suff.	0			Ind. Ind.	0	0	Cold, illness.	0	L. base.	Sev. till D.	till D.	D. 4th d.	—	2
336 "	F.	19	Tot. abs. Suff.	Pneum.	1		Ind. Ind.	0	0	Chilliness.	0	L. base.	Sev. 7 d. Grad.	12 d.	R.	—	3
337 E. Long Fox, M.D.	M.	22	Temp. Suff.	Pneum. Erysip.		6 1	Good. Good.	0	<i>L. pneum.</i> 5 y. ago, <i>L. pleuro-</i> <i>pneum.</i> 2 y. ago.	Malaise, pain in side.	r.	R. base, L. whole	Mod. 16 d. Grad.	24 d.	R.	—	
338 "	M.	19	Temp. Suff.	Pneum. Tonsill. Bron. Cat.		6 4 12	Good. Ind.	0	0	Headache, oppression of chest.	r.	R. base to apex.	Mod. 6 d. Sud.	10 d.	R.	0	
339 "	F.	54	Temp. Suff.	Pneum. Tonsill. Bron. Cat.		6 4 12	Good. Good.	0	0	Malaise 2 d.	r.	R. base. L. whole	Sev. 14 d. Grad.	17 d.	R.	0	
340 T. F. Raven, L.R.C.P.	F.	35	Intemp. Insuff.	Enteric. Herpes.		1 1	Good. Good.	0	Angular curv., bron. cat., alcoholism 2 y. ago.	Catarrh, debility.	0	R. base.	Mod. till D.	till D.	D. 7th d.		4
341 Tom Robinson, M.D.	F.	74	Temp. Suff.	Tonsill. Bron. Cat. Erysip. Scarlat.		2 + 1 +	Ind. Good.	0	Double aortic mur.	Headache, aching of limbs.	r.	R. base.	Mod. 6 d. Sud.	6 d.	R.	Slight cough, great weakness.	
342 "	M.	62	Temp. Suff.	0			Ind. Ind.	0	Bronch.	Vomiting, hesitance.	r.	L. base.	Sev. till D.	till D.	D. 4th d.		5
343 "	M.	41	Intemp. Suff.	Tonsill. Bron. Cat.		+	Good. Good.	0	Organic stricture. 2nd year	Vomiting.	r.	Both bases.	Mod. till D.	till D.	D. 8th d.		





Observer's Name.	Sex.	Ago.	Temperate or other- wise. Food.	Concurrent cases of illness in	Same house.	Same district.	Sanitary state of house " " of district	Family history of lung disease.	Previous illnesses of patient.	Premunitory symptoms.	Mode of onsch.	Part of lungs affected.	Character Duration Termination of fever.	Duration of physical signs.	Result.	Sequelae.	Note.
355 J. Aikman, M.D.	F.	76	Temp. Suff.	—	—	—	Good. Good.	2 S. d. phth.	Slight gout.	Dyspnoea, weakness.	r.	R. whole L. base.	Mod. till D.	till D.	D. 4th d.	—	1
356 "	F.	6	Tot. abs. Suff.	Scarlat. within 50 yds.	1	4	Good. Ind.	1 U. chr. phth.	0	Anorexia, dyspnoea, R. decubi- tus.	r.	R. base.	Sev. 4 d. Sud.	8 d.	R.	—	
357 J. Mackenzie Booth, M.D.	M.	52	Temp. Suff.	Tonsill. Bron. Cat.	1	+	Ind. Ind.	0	<i>Pneum.</i> twice, last 3 y. ago.	Nasal catarrh.	r.	R. base.	Sev. till D.	till D.	D. 7th d.	—	2
358 "	F.	20	Tot. abs. Suff.	Bron. Cat.	1	+	Good. Ind.	F. pneum.	<i>Pneum.</i> 5 y. ago Bron. 2 y. ago.	Chilliness, malaise.	r.	R. base.	Mod. 7 d. Grad.	9 d.	R.	—	
359 S. H. Burton, M.B.	M.	40	Temp. Suff.	0	—	—	Ind. Ind.	1 B. 36, double pneum. at same time.	0	Malaise.	r.	Both bases.	Mod. 12 d. Grad.	14 d.	R.	—	
360 A. S. Currie, M.D.	M.	—	Temp. Suff.	0	—	—	Good. Good.	—	<i>Pneum.</i> 6 y. ago.	Pain in side, dry skin.	r.	R. whole	Sev. 7 d. Grad.	—	R.	—	
361 "	F.	41	Temp. Suff.	Bron. Cat.	—	+	Good. Good.	—	Leucocythoe- mia.	Malaise, cough, pain in back.	r.	L. base.	Sev. till D.	—	D.	—	
362 D. Arthur Davies M.B.	F.	18	Temp. Suff.	Pneum. Tonsill. Bron. Cat.	1	2 3	Good. Good.	M. ac. pneum.	0	Catarrh, pain R. side.	r.	Both bases.	Sev. 4 d. Sud.	7 d.	R.	0	
363 T. V. de Denne, L.R.C.P.	M.	48	Temp. Suff.	Pneum. Measles. Pertussis. Scarlat.	2	+	Ind. Ind.	M. bron.	0	0	r.	R. base. L. whole	Sev. till D.	till D.	D. 37th d.	—	3
364 C. E. Douglas, M.D.	M.	5	Tot. abs. Suff.	—	—	—	Ind. Ind.	0	0	Pain and tenderness over liver, vomiting.	0	R. base.	Sev. 7 d. Sud.	—	R.	0	

365	M.	49	Temp. Suff.			Ind. Ind.	Dyspnoea.	General, but esp. at joints.	r.	A. base.	Stom.	U.	14.	Examination.
366 F. H. Drake, L.R.C.P.	M.	33	Temp. Suff.	Catarrh. Tonsill. Bron. Cat. Parotitis. Rheum.	1 1 1 1 1	Good. Good.	0	Malaise, pain in joints 3 d.	r. R. apex.	8 d.	Sev. 10 d. Sud.	— Grad.	R.	—
367 J. T. Faulkner, M.B.	M.	37	Temp. Suff.	Pneum. Bron. Cat. Erysip.	3 3 2	Ind. Ind.	Bell's palsy 6 m. ago, bron. 4 m. ago.	Muscular pain, malaise.	0 R. base.	30 d.	Sev. 8 d. Sud.	R.	R.	Rheumatic pains on going out.
368 S. Winter-Fisher, M.D.	F.	49	Temp. Suff.	—		Good. Good.	0	Pain R. side.	0 R. base.	15 d.	Mod. 10 d. Grad.	R.	R.	0
369 W. Frew, M.B.	M.	6	— Suff.	Pneum. Enteric. Catarrh. Tonsill. Bron. Cat. Erysip.	1 2 1 1 1 2	Good. Good.	G. F. d. "de-eline," S. pneum.	Headache, fever, pain in side.	0 R. apex.	9 d.	Sev. 5 d. Sud.	R.	R.	0
370 G. A. Gibson, M.D.	M.	2	Tot. abs. Suff.	Pneum. Measles. Scarlat. Catarrh. Tonsill. Erysip.	1 6 4 10 1	Good. Good.	1 B. and 1 S. ac. pneum. while teething.	Fever, pain, anorexia.	r. L. base.	18 d.	Mod. 10 d. Grad.	R.	R.	0
371 P. F. Graham, M.D.	M.	48	Temp. Suff.	Pneum. Typhus.	3 +	Good. Ind.	Pneum. R. base 10 y. ago.	Great thirst.	r. R. base.	22 d.	Sev. 10 d. Grad.	R	R	0
372 G. Hunter, M.D.	M.	40	Intemp. Suff.	0		Ind. Ind.	0	Prolonged vomiting.	r. R. whole L. base.	till D. 9th d.	Sev. till D.	D. 9th d.	D.	
373 Hugh R. Ker, F.R.C.S.	M.	25	Temp. Suff.	—		Bad. Bad.	F. d. chr. bron.	0	r. Both bases.	21 d.	Sev. 14 d. Grad.	R.	R.	0

1. Albumin. and granular casts, not known before attack. 2. Just left off flannel clothes. 3. After 5th d. much relieved till 20th d., then fresh symps. beginning R. base and spreading over both lungs. 4. On 11th d. coughed up complete cast of bronch. tubes to about 4th division: now in Leeds Museum. 5. Closely resembled attacks of brother and sister. 6. Complicated by typhoid symps., diarrhoea, and vomiting.



Observer's Name.	Sex.	Age.	Temperate or other-wise. Food.	Concurrent cases of illness in	Same house.	Same district.	Sanitary state of house " " of district	Family history of lung disease.	Previous illnesses of patient.	Premontory symptoms.	Mode of onset.	Part of lungs affected.	Character of fever. Duration of fever.	Duration of physical signs.	Result.	Sequelæ.	Note.
374 Hugh R. Ker, F.R.C.S.	F.	54	Temp. Suff.	0			Ind. Ind.	0	0	Diarrhoea, vomiting.	r.	R. base.	Mod. 10 d. Grad.	15 d.	R.	0	1
375 "	M.	16	Temp. Suff.	0			Bad. Bad.	0	0	0	r.	L. whole nearly.	Sev. 10 d. Grad.	15 d.	R.	0	
376 "	M.	42	Temp. Suff.	Pneum. Bron. Cat.		1 1	Good. Ind.	0	0	0	r.	L. whole	Sev. 7 d. Grad.	10 d.	R.	0	
377 H. Kershaw, M.R.C.S.	M.	19	Temp. Suff.	Pneum. Catarrh. Tonsill. Bron. Cat. Scarlat.		2 1 1 +	Ind. Bad.	0	0	0	r.	R. base.	Sev. 9 d. Grad.	15 d.	R.	0	2
378 "	M.	18	Temp. Suff.	Pneum. Scarlat. Bron. Cat.		1 1 +	Ind. Ind.	0	0	0	r.	L. base.	Mod. 6 d. Grad.	10 d.	R.	0	
379 Robert Kirk, M.D.	M.	41	Tot. abs Suff.	Pneum. Tonsill. Herpes. Bron. Cat.		3 4 1 +	Good Good.	Ch. d. phth.	0	0	r.	Both bases.	Sev. 12 d. Grad.	17 d.	R.	0	
380 W. Lamb, M.D.	M.	81	Temp. Suff.	Pneum. Measles. Bron. Cat.		1 1 6	Ind. Ind.	2 Sons d. phth. when young men.	0	Chill, cough, hemopt.	0	R. base.	0	Over 20 d.	R.	0	
381 Alex. Mac- donald, L.R.C.P.	F.	12	Tot. abs. Suff.	Catarrh. Tonsill. Bron. Cat.		+	Good. Good.	0	0	0	r.	R. base.	Sev. 7 d. Sud.	11 d.	R.	—	
382 W. H. Mason, M.R.C.S.	M.	27	Temp. Suff.	Pneum. Measles. Enteric. Catarrh. Bron. Cat. Erysip.		2 3 3 +	Good. Good.	1 B. d. phth. 31.	0	Pain in side.	r.	R. whole	Sev. till D.	till D. 11th d	D.		

384 A. D. Leith Napier, M.D.	F	17	Tot. abs. Suff.	Pneum. Scurlat. Enteric. Measles. Catarrh. Bron. Cat.	3 2 2 3 2 2	Good. Good	1 R. d. pneum. F. bron. and asthma.	0	Langnor, anorexia	r.	R. whole L. base.	15 d. Grad.	R.	—
385 James Neil, M.D.	F.	70	? Insuff.	0	Good. Ind.	0	0	0	Fever, slight pain under scapula.	0	R. apex.	Mod. till D.	D. 6th d.	3
386 C. V. Newstead, M.R.C.S.	M.	26	Temp. Suff.	—	Good. Good.	0	0	0	0	r.	R. base.	Sev. 10 d. Sud.	R.	0
387 R. P. Oglesby, M.D.	M.	35	Intemp. Suff.	Pneum. Enteric.	3 2	Bad. Ind.	1 S. d. phth. 1 Cousin do.	<i>Pleuro-pneum.</i> 7 y. ago.	Vomiting, anorexia.	r.	L. base.	Sev. 21 d. Grad.	R.	0
388 "	M.	42	Intemp. Suff.	Pneum. Enteric.	3 3	Good. Good.	0	0	Headache, vomiting.	r.	L. base.	Sev. 21 d. Grad.	R.	—
389 Thos. J. Oller- head, L.R.C.P.	F.	30	Temp. Suff.	Catarrh. Tonsill. Bron. Cat.	+	Good. Good.	0	<i>Pleuro-pneum.</i> 15 y. ago.	Fever.	0	Both bases.	Sev. 12 d. Grad.	R.	—
390 C. A. Owens, M.D.	F	15	Temp. Suff.	Pneum. Bron. Cat.	7 +	Good. Good.	F. pneum. 5 y. ago. 1 B. pneum. 1 y. ago. 2 S. pneum. 1 y. ago.	0	Headache, malaise, catarrh.	r.	R. base.	Sev. 7 d. Grad.	D. 11th d	
391 Lawrence Phil- lips, M.R.C.S.	F.	11	Tot. abs. Suff.	Pneum. Fever. Tonsill. Bron. Cat.	2 1 1 7	Good. Good.	2B. } d. bron. 1S. } F. "has one lung."	Delicate.	Pain in side, cough, langnor.	0	Both bases.	Mod. 6 d. Sud.	R.	—
392 A. W. Mayo Rob- son, F.R.C.S.	F	2	Temp. Suff.	—	Good. Good.	0	0	0	—	r.	L. base.	Sev. 6 d. Sud.	R.	0

1. Complicated by typhoid symps., diarrhoea and vomiting. 2. Sev. epistaxis 3rd. d. 3. During attack of acute mania. Expos. to cold. 4. Many days before signs appeared, patient had continued vomiting, vomit unbearably offensive; no symp. of abscess in lung, recov. very slow. 5. Acute gout supervened during attack; sudden swelling of L. leg from thrombosis; recov. not satisfact.

Observer's Name.	Sex.	Age.	Temperate or otherwise. Food.	Concurrent cases of illness in	Same house.	Same district.	Sanitary state of house " " of district	Family history of lung disease.	Previous illnesses of patient.	Premontory symptoms.	Mode of onset.	Part of lungs affected.	Character of fever. Duration Termination	Duration of physical signs.	Result.	Sequelæ.	Note.
393 A. W. Mayo Robinson, F.R.C.S.	M.	70	Temp. Suff.	Pneum. Measles. Tonsill. Bron. Cat. Erysip.	2 + 2 1	2	Good.	0	0	—	r.	L. base.	Sev. till D.	till D.	D. 7th d.		
394 "	F.	62	Temp. Suff.	Fever. Catarrh. Tonsill. Bron. Cat.	4 1 1 8	4	Good.	0	—	Pain in side.	r.	L. apex.	Mod. till D. 7 d. Sud.	till D. 17th d	D. 17th d		1
395 A. H. Robinson, M.D.	M.	24	Temp. Suff.	Pneum. Catarrh. Tonsill. Bron. Cat.	3 12 12	3	Bad. Bad.	0	0	Malaise, headache, pain inside.	r.	L. base.	Mod. 7 d. Grad.	14 d.	R.	Pain in side	
396 Thos. R. Ronaldson, M.D.	F.	2	Tot. abs. Suff.	Tonsill. Bron. Cat.	+	+	Ind. Good.	0	0	Cold.	0	L. apex.	Sev. 7 d. Sud.	11 d.	R.	0	
397 "	F.	8	Tot. abs. Suff.	Pneum. Scarlat. Tonsill. Bron. Cat.	2 3 +	2	Good. Good.	F's F. d. phth.	Measles 3 y. ago.	0	r.	Both bases.	Sev. 7 d. Sud.	14 d.	R.	0	2
398 "	M.	10	Tot. abs. Suff.	Pneum. Scarlat. Tonsill. Bron. Cat.	2 few. +	2	Good. Good.	F's M. d. pneum. F's B. d. phth.	Scarlat. Measles. Variocella.	Pain, dyspnoea.	0	L. base.	Mod. 7 d. Sud.	13 d.	R.	0	
399 E. T. Tylecote, M.D.	M.	24	Temp. Suff.	—			Good. Good.	M. d. phth.	Diphther. 4 y. ago.	0	r.	Both bases.	Mod. 7 d. Grad.	14 d.	R.	0	3
400 W. E. W. Vaughan, M.R.C.S.	M.	40	Temp. Suff.	Pneum. Catarrh. Herpes. Erysip.	2 2 2 1	2	Ind. Bad.	0	0	0	r.	R. base.	Sev. 10 d. Sud.	14 d.	R.	0	
401 J. O. Wilson, M.D.	M.	23	Temp. Suff.	Pneum. Pertussis.	1 +	1	Good. Good.	0	Occas. gravel colic	Pains.	r.	R. base.	Sev. 8 d.	14 d.	R.	0	



403 W. Snodden, M.D.	M.	45	Temp. Suff.	Tonsill. Herpes. Bron. Cat.	2 1 3	Ind. —	B. d. pneum.	Pneum. 9 m. ago.	Malaise 2 d.	0	R. base. L. base.	Sev. 7 d. Sud.	9 d.	R.	0	
404 James Barr, L.R.C.P.	M.	25	Intemp. Suff.	0		Good. Good.	0	0	Malaise 1 w.	r.	R. whole L. base.	Sev. 13 d. Sud.	17 d.	R.	0	
405 "	F.	16	Tot.abs. Suff.	Pneum.	2	Good. Good.	F. d. pneum. 38.	0	Malaise.	r.	Both bases.	Sev. 14 d. Sud.	18 d.	R.	0	
406 "	F.	14	Tot.abs. Suff.	0		Good. Good.	0	0	0	r.	L. apex.	Sev. 8 d. Sud.	11 d.	R.	0	
407 G. W. Beattie, M.D.	M.	40	Temp. Suff.	Pneum. Scarlat.	3 1	Ind. Ind.	1 B. } d. phth. 2 S. } 1 Ch. d. men- ingit. 1 Ch. d. pneum. 2 Ch. bron.	0	Langnor many w.	r.	R. base.	Sev. till D.	till D. 4th d.	D.		4
408 G. A. Brown, M.R.C.S.	M.	50	Temp. Suff.	Pneum.	1	Good. Good.	0	Bronch. 4 y. ago, empty- senia.	0	r.	Both bases.	Sev. till D.	till D. 8th d.	D.		5
409 John Brown, L.R.C.P.	M.	31	Tot.abs. Suff.	Pneum. Catarrh. Bron. Cat. Erysip.	3 2 9 1	Ind. Mxd.	F. d. phth. 35 1 S. phth.	0	Catarrh 3 d.	r.	R. base.	Sev. 10 d. Grad.	12 d.	R.	Debility.	6
410 W. R. Buckell.	M.	—	— Suff.	Pneum. Scarlat. Catarrh. Tonsill.	2 3 3 1	Good. Good.	0	—	Catarrh.	0	R. base.	Mod. 10 d. Grad.	—	R.	—	
411 T. J. Burroughs, M.D.	F.	11	Tot.abs. Suff.	Pneum.	2	—	0	0	0	r.	R. base.	Sev. 9 d. Grad.	14 d.	R.	—	

1. Convalescent; D. by apoplexy. 2. Expos. to cold and wet. 3. Rigor while driving in a snowstorm. 4. Seemed fatal from onset. 5. Daughter in same house, granddaughter a few doors off, had pneum. at same time. 6. Exp. to draught.

Observer's Name.	Sex.	Age.	Temperate or other-wise. Food.	Concurrent cases of illness in	Same house.	Same district.	Sanitary state of house " " of district	Family history of lung disease.	Previous illnesses of patient.	Premontory symptoms.	Mode of onset.	Part of lungs affected.	Character of fever. Duration Termination	Duration of physical signs.	Result.	Sequelæ.	Note.
412 T. J. Burroughs, M.D.	M.	44	Temp. Suff.	Pneum. Catarrh.		2	Ind. —	1 B. d. phth.	0	—	—	R. base.	Sev. 8 d. Grad.	12 d.	R.	—	1
413 Charles W. Chaldecott, M.R.C.S.	F.	47	Temp. Suff.	0			Good. Good.	0		Malaise.	r.	R. base. L. whole	Sev. till D.	till D. 10th d	D.	—	
414 C. P. Coombs, M.D.	M.	33	Temp. Suff.	Pneum. Measles. Rötheln. Catarrh. Tonsill. Bron. Cat. Erysip.		3 + + + + + +	Ind. Good.	M. d. lung disease.	Bronch.	Catarrh.	r.	L. base.	Sev. 8 d. Sud.	Over 21 d.	R.	0	
415 "	M.	57	Temp. Suff.	Pneum. Catarrh. Tonsill. Herpes. Bron. Cat. Erysip.		6 + + + + +	Ind. Good.	0	0	Catarrh.	r.	L. base.	Mod. — Sud.	7 d.	R.	0	
416 D. Arthur Davies, M.B.	F.	17	Temp. Suff.	Pneum. Measles. Tonsill.		2 + 1	Good. Good.	0	0	0	r.	R. base.	Sev. 6 d. Sud.	12 d.	R.	0	
417 "	M.	38	Intermp. Suff.	Pneum. Measles. Bron. Cat.		1 3 2	Good. Good.	0	Subac gout occas.	Knocked up.	r.	R. apex.	Mod. 6 d. Sud.	4 d.	R.	0	2
418 G. H. Davis, L.R.C.P.	M.	49	Temp. Suff.	Pneum. Diphth. Herpes.	1	2 1	Good. Good.	—	0	Cold.	r.	R. whole L. base.	Sev. 12 d. Grad.	—	R.	0	
419 Edward Drummond, M.D.	M.	41	Temp. Suff.	0			Good. Ind.	F. d. fibroid phth.	Bronch.	0	r.	Both bases.	Mod. 7 d.	10 d.	R.	0	3





Observer's Name.	Sex.	Age.	Temperate or otherwise. Food.	Concurrent cases of illness in	Same house.	Same district.	Sanitary state of house " " of district	Family history of lung disease.	Previous illnesses of patient.	Premontory symptoms.	Mode of onset.	Part of lungs affected.	Character of fever. Duration Termination	Duration of physical signs.	Result.	Sequelæ.	Note.
430 J. Johnston, M.D.	M.	22	Temp. Suff.	Pneum.		1	Ind. Ind.	U. pneum.	0	0	r.	R. base.	Mod. 7 d. Grad.	12 d.	R.	0	
431 "	M.	28	Temp. Suff.	—			Ind. Ind.	—	0	0	r.	R. base.	Sev. 8 d. Grad.	14 d.	R.	0	
432 J. W. Lane, M.D.	M.	18	Temp. Suff.	Pneum.	4		Ind. Ind.	0	—	Cough, pain in chest.		Both bases.	Sev. 14 d. Grad.	—	R.	0	
433 "	M.	16	Temp. Suff.	"			" "	0	Diseases of childhood.	Cough, thirst.	r.	Both bases.	Sev. — Grad.	—	R.	0	
434 "	F.	10	Temp. Suff.	"			" "	0	Diseases of childhood.	Cough, thirst.	r.	R. base.	Sev. 21 d. Grad.	—	R.	Cough, dul- ness over R. lung.	
435 "	M.	7	Temp. Suff.	"			" "	0	Measles 1 y. ago.	—	—	Both bases.	Mod. 10 d. Grad.	—	R.	0	
436 "	M.	2	Temp. Suff.	"			" "	0	Measles 1 y. ago.	Cough.	—	Both bases.	Mod. 10 d. Grad.	—	R.	0	
437 "	M.	46	Temp. Suff.	Pneum.		3	Bad. Bad.	0	—	Cough.	r.	Both bases.	Mod. 10 d. Grad.	16 d.	R.	0	
438 "	M.	42	Temp. Suff.	Pneum.		3	Ind. Ind.	0	—	Dyspnoea.	r.	Both bases.	Sev. 14 d. Grad.	30 d.	R.	—	1
439 "	M.	17	Temp. Suff.	Pneum.		3	Ind. Ind.	0	0	Cough.	r.	Both bases.	Sev. 10 d. Sud.	14 d.	R.	0	
440 A. W. Leachman, M.D.	M.	43	Intemp. Suff.	—			Ind. Ind.	B. d. plith. 36. Ind. of lungs 7 y.		0	r.	L. base.	Mod. 9 d.	Over 10 d.	R.	Chronic swollen	2

	F.	39	Temp. Suff.	Tonsill. Bron. Cat. Erysip.	+ +	Bad. Good.	B. " 1 ago	0	Pneum. 10 y. ago.	0	Both aplees.	Sud. Mod. 10 d. Grad.	14 d.	R.	0
442 W. E. Linscombe, L.R.C.P.															
443 "	M.	49	Temp. Suff.	Tonsill. Bron. Cat.	5 8	Good. Good.	0	0	0	Cough.	0	R. base. 10 d. Sud.	15 d.	R.	Muse. rheu- matism of calf. 0
444 A. M. McAl- dowie, M.D.	F.	19	Temp. Suff.	Pneum. Measles. Bron. Cat. Erysip.	2 14 2	Good. Good.	0	0	Ac. rheum. twice.	Pain in side.	r.	L. base. 12 d. Grad.	21 d.	R.	0
445 "	F.	51	Temp. Suff.	Catarrh. Tonsill. Erysip.	3 4 1	Good. Good.	F. d. bronch. 55	Bronch.	Bron. Cat.	0	0	R. base. 16 d. Grad.	21 d.	R.	—
446 A. W. Macfar- lane, M.D.	F.	26	Temp. Suff.	Pneum. Dysentery. Catarrh. Tonsill. Bron. Cat.	4 4 3 2 4	Good. Good.	—	Bronch.	0	0	r.	R. base. 11 d. Grad.	22 d.	R.	0
447 "	M.	6	Tot. abs. Suff.	Pneum. Dysent. Catarrh. Tonsill. Bron. Cat.	4 4 2 1 6	Ind. Good.	—	Pneum. 4 y. ago.	0	0	0	L. base. 6 d. Sud.	12 d.	R.	0
448 Duncan J. Mac- kenzie, M.D.	M.	19	Tot. abs. Suff.	Pneum. Febric. Tonsill. Herpes. Bron. Cat.	1 2 1 1 9	Good. Ind.	M. } d. plith. B. }	Typhilitis, nose rheu- matism.	Nausea, vomiting.	r.	L. base. 5 d. Grad.	18 d.	R.	0	
449 J. A. Mackenzie, M.B.	M.	18	Temp. Suff.	Pneum. Scarlat. Tonsill. Bron. Cat. Erysip.	2 1 1 3 1	Good. Ind.	0	0	0	Chills, vomiting.	r.	R. base. 5 d. Sud.	9 d.	R.	0
450 "	M.	18	Temp. Suff.	Pneum. Scarlat. Tonsill. Bron. Cat.	2 2 2 2	Good. Good.	1 S. pneum.	Diseases of childhood.	Malaise, sudden pain.	0	L. base. 4 d. Sud.	18 d.	R.	0	

1. Valvular disease and albuminuria. 2. Went to work 4 m. after onset; ten m. after onset quite healthy, chest normal. 3. Cardiac hypertrophy, mitral disease.

Observer's Name.	Sex.	Age.	Temperate or other-wise. Food.	Concurrent cases of illness in	Same house.	Same district.	Sanitary state of house " " of district	Family history of lung disease.	Previous illnesses of patient.	Premontory symptoms.	Mode of onset.	Part of lungs affected.	Character of fever Duration of physical signs.	Result.	Sequelæ.	Note.
451 W.A.M'Lachlan, M.D.	M.	23	Temp. Suff.	Pneum. Tonsill. Herpes. Erysip.	1	1 1 1 2	Ind. Ind.	0	0	0	r.	L. base.	Sev. 7 d. Grad.	R.	0	
452 "	M.	39	Temp. Suff.	Pneum. Enteric. Bron. Cat.	1	3 2	Ind. Ind.	0	0	0	r.	R. base.	Mod. 11 d. Grad.	R.	0	
453 "	F	46	Temp. Suff.	Enteric. Diphth. Herpes. Bron. Cat.	1	3 3 1	Good. Ind.	0	0	Gastro- intestinal derange- ment.	0	R. base.	Mod. 10 d. Grad.	R.	0	1.
454 A. Cowley Malley, M.B.	M.	22	Temp. Suff.	Tonsill. Bron. Cat.	1	3 5	Ind. Good.	0	0	Bron. 2 m.	r.	L. apex.	Sev. 7 d. Sud.	R.	0	
455 Thomas Martin, L.R.C.P.	M.	70	Temp. Suff.	Pneum. Herpes. Bron. Cat. Erysip.		3 2 7 1	Ind Good.	Sou d. phth.	0	0	r.	L. base.	Mod. 7 d. Grad.	R.	0	
456 "	F.	80	Temp. Insuff.	Pneum. Herpes. Bron. Cat.		3 2 7	Ind. Good.	0	0	0	r.	R. whole	Mod. till D.	D. 6th d.		
457 "	M.	30	Temp. Suff.	Pneum. Herpes. Bron. Cat.		3 2 7	Good. Good.	—	0	0	r.	L. base.	Mod. 6 d. Grad.	R.	Chest weak 2 m.	
458 T. Frederick Pearse, M.D.	M.	11	Temp. Suff.	0			Ind. Ind.	?	?	Delirium, fever.	r.	L. base.	Sev. 8 d. Sud.	R.	0	
459 C. H. Phillips, M.R.C.S.	F.	32	Tot. abs. Insuff.	Pneum.		4	Ind. Ind.	F. d. phth. 58 1 S. " 38	0	—	r.	R. base.	Sev. 10 d.	D. 35th d.		2

461	"	M.	5½	Temp. Insuff.	Pneum. Enteric. Herpes.	1 3 3 4	Good. Ind.	F's S. d. phth. M's 2 B. "	Mauslos 2 y. ago.	ache. caracho. Drowsiness.	?	L. whole	Sev. 14 d. Sud.	18 d.	R.	Weakness.	4
462	"	F.	1	Tot. abs. Insuff. (Suck.)	Pneum. Enteric. Herpes.	2 2 2 2	Good. Ind.	0	Per- tussis last measles } 6 m. cough since.	Debility.	?	R. apex	Mild. 6 d. Sud.	11 d.	R.	Cough persists.	
463	"	F.	7	Tot. abs. Suff.	Pneum. Tonsill. Bron. Cat.	3 1 2	Good. Good.	M's S. d. phth. M's F's fam. phth.	Measles 4 y. ago. Pertuss. 5 y. ago.	0	r.	L. apex.	Mod. 4 d. Sud.	8 d.	R.	--	
464	"	M.	2½	Tot. abs. Suff.	Pneum. Enteric. Herpes. Bron. Cat.	3 3 3 +	Good. Ind.	M's fam. phth. and asthma. F. bron.	0	Sickness.	?	L. apex. to base.	Mod. 26 d. Sud.	31 d.	R.	Debility, anemita, cough.	5
465	"	F.	14m	Tot. abs. Insuff. (Suck.)	Pneum. Tonsill. Bron. Cat.	3 2 2	Ind. Ind.	0	Measles, } 6 m. Pertuss. } ago. Cough since, rickets.	--	?	R. apex.	Mod. 7 d. Sud.	9 d.	R.	Cough.	
466	James Reid, F.R.C.S.	M.	10	Temp. Suff.	Pneum. Mumps.	1 6 +	Good. Good.	M. d. phth. 4 B. and S. d. hydroceph.	Anemia 1 y. ago. Delicate.	0	0	R. base.	Sev. 6 d. Sud.	over 30 d.	R.	Chron. pneum.	6
467	"	M.	12	Tot. abs. Suff.	Ditto.		Good. Good.	M's M. d. phth. M's fam. phth. 1 B. pneum.	--	Catarrh 6 d.	0	R. base.	Mod. 8 d. Sud.	over 27 d.	R.	--	7
468	"	M.	23	Temp. Suff.	0		Good. Good.		Pneum. R. L. S. y. ago.	Depression, chilliness.	r.	L. base.	Sev. 8 d. Grad.	14 d.	R.	0	
469	Charles J. Renshaw, M.D.	M.	60	Temp. Suff.	Pneum. Scarlat. Catarrh. Tonsill. Herpes. Bron. Cat. Erysip.	1 3 + 6 4 4 6 6 2	Good. Good.	F. } d. chr. M. } bron.	Pneum. sever- al attacks.	Fever, cough.	r.	Both. bases.	Sev. 2 d. Grad.	21 d.	R. part.	Chron. pneum., asthmatic breathing.	8

1. Was in bed with broken leg. 2. Miscarriage at 6th m. soon after onset; rapid emaciation, yellow sputum, night sweats. 3. Four cases of acute pneum. recently occurred in a very small area where there has been offensive smell from pigstyes. 4. Dulness first at lower edge of upper lobe, then apex, then base. 5. Mucous and bloody stools; case was, however, decidedly not one of enteric fever. 6. Mumps on 7th d. 7. Mumps on 5th d. 8. Pleurisy R. base 3 w. later; D. on 3rd d.



Observers	Sex.	Age.	Temperate or other-wise. Food.	Concurrent cases of illness in	Same house.	Same district.	Sanitary state of house " " of district	Family history of lung disease.	Previous illnesses of patient.	Prenatal symptoms.	Mode of onset.	Part of lungs affected	Character of fever. Duration of physical signs.	Result.	Sequelæ.	Note.
470 J. Bruce Ronaldson, L.R.C.P.	F.	12	Tot. abs. Suff.	Pneum. Tonsill. Bron. Cat.	1	3 6 6	Good. Good.	0	—	Malaise.	r.	R. base.	Sev. 5 d. Sud.	R.	0	1
471 E. S. Scott, M.B.	M.	18	Tot. abs. Suff.	Tonsill. Bron. Cat. Erysip.		1 3 1	Ind. Ind.	F. bron.	<i>Pneum.</i> 6 m. ago.	0	r.	R. apex.	Sev. 8 d. Sud.	R.	Mental depression.	
472 "	M.	39	Temp. Suff.	0			Good. Good.	0	Bron. 2 y. ago. Cough, } 2m dyspnoea } ago	Cough, short breath.	r.	L. whole	Mod. 10 d. Grad.	R.	0	
473 J. E. Smith, M.D.	M.	17	Temp. Suff.	Scarlat.		+	Ind. Ind.	F. lung disease.	—	Pain, fever, headache.	—	L. base.	Sev. 13 d. Grad.	R.	Rheumatic pains in joints.	
474 W. Shaw, M.D.	M.	29	Temp. Suff.	Tonsill. Bron. Cat.		1 3	Good. Ind.	0	0	Cold, fever.	r.	Both bases.	Mod. 5 d. Sud.	R.	—	
475 Herbert Sloman, L.R.C.P.	F.	3	Temp. Suff.	Pneum. Diphth. Measles. Catarrh. Tonsill. Herpes. Bron. Cat. Erysip.	2 1 1	20 6 5 4 12 9	Bad. Ind.	0	0	Sickness, anorexia.	0	R. apex.	Sev. 6 d. Grad.	R.	0	2
476 "	F.	27	Temp. Suff.	Pneum. Diphth. Measles. Tonsill. Bron. Cat. Erysip.		20 6 5 4 12 9	Ind. Ind.	1 S. ineip. phth. F. asth. and bron.	Ac. rheum. <i>R. pneum.</i> 2 y. ago.	Debility, cough some w.	r.	R. whole	Mod. 7 d. Sud.	R.	0	
477 "	M.	67	Temp. Suff.	Tonsill. Bron. Cat. Erysip.	1	12 9	Ind. Ind.	0	<i>Pneum.</i> 20 y. ago, asthma since.	Malaise, bronch. catarrh.	r.	R. whole	Sev. 10 d. grad.	R.	Rheumatic pains.	
478 "	M.	8	Temp. Suff.				Ind. Fubr.	2 U. } d. phth. 1 A. }	Ac. bron. 2 y. ago. Frequent	Malaise, aching	0	R. apex. L. base.	Sev. 7 d.	R.	0	

No.	Sex	Age	Date	Temp.	Pulse	Respiration	Blood	Stool	Urine	Sweat	Skin	Mucous	Nervous	General	Remarks	Outcome
480	"	M.	6	Insuff.	6	13	Bad.	0	0	0	0	0	0	0	0	0
481	"	M.	70	Temp. Insuff.	13	3	Bad.	0	0	0	0	0	0	0	0	0
482	S. Sloman, jun. L.R.C.P.	M.	45	Intemp. Suff.	13	3	Bad.	0	0	0	0	0	0	0	0	0
483	A. Stewart, M.D.	F.	21	Temp. Suff.	13	3	Bad.	0	0	0	0	0	0	0	0	0
484	"	F.	20	Temp. Suff.	13	3	Bad.	0	0	0	0	0	0	0	0	0
485	"	M.	12	Temp. Suff.	13	3	Bad.	0	0	0	0	0	0	0	0	0

1. B. said to have had chest disease, followed by ac. mania with which he died in asylum. 2. Onset, 3.6.83. 1 B. (4t) attacked with pneum. 5.6.83. 1 S. (6) ditto, 8.6.83. F. lumbago and cold, 8.6.83. 3. Crisis 6th d., relapse 7th d. extension of consolidation. Wife d. pleuro-pneum. 21 d. before his attack. Son pleuro-pneum. 4 d. before, entirely nursed by his F.

Observer's Name.	Sex.	Age.	Temperate or otherwise. Food.	Concurrent cases of illness in	Same house.	Same district.	Sanitary state of house " " of district	Family history of lung disease.	Previous illnesses of patient.	Premontory symptoms.	Mode of onset.	Part of lungs affected.	Character of fever. Duration Termination	Duration of physical signs.	Result.	Sequelae.	Note.
486 C. E. Underhill, M.B.	F.	24	Temp. Suff.	0			Good. Good.	0	Scarlat. of inflam. of bowels thrice, once severely.	Malaise, chilliness, pain in side.	r.	Both bases.	Sev. 21 d. Grad.	Over 60 d.	R.	—	1
487 J. G. U. West, L.R.C.P.	F.	27	Temp. Suff.	Pneum. Enteric. Catarrh. Bron.Cat.		3 2 1 1	Ind. Ind.	F. d. bronch. asthma 48.	0	Bronch. 3 d.	r.	R. base.	Mod. 10 d. Sud.	14 d.	R.	0	2
488 W. White, M.D.	M.	42	Temp. Suff.	Pneum. Enteric. Catarrh. Bron. Cat. Erysip.	1 1	3 3 + +	Good. Ind.	1 B.d.hæmopt. 1 cousin d. phth.	0	Cough.	r.	L. base.	Mod. 16 d.	till D.	D. 20th d	—	3
489 "	F	36	Temp. Suff.	Ditto.			Good. Good.	0	Double pneum. 8 y. ago, delicate.	Cough.	r.	R. base.	Mild 8 d. Sud.	21 d.	R.	—	—
490 "	M.	37	Intemp. Suff.	Ditto.			Good. Ind.	0	—	Catarrh 3 d.	r.	L. base.	Mod. 9 d. Sud.	14 d.	R.	—	—
491 "	M.	28	Temp. Suff.	Pneum. Catarrh. Tonsill.		5 2 2	Good. Good.	0	Broncho-pneum. sev. 2 y. ago.	—	—	R. base.	Sev. till D.	till D.	D. 8th d.	—	4
492 G. G. Whitwell, M.B.	M.	50	Intemp. Suff.	Bron. Cat. Erysip.		1 1	Ind. Ind.	F. inflam.phth. 5 B. and S. (of 10) d. phth.	Coughs and colds.	Bronch. 7 d.	—	R. mid. L. base.	Sev.	—	D. 8th d.	—	—
493 C. E. Abbott, L.K.Q.C.P.	F.	9	Tot. abs. Suff.	—			Ind. Ind.	F. d. phth.	Scarlat. 5 m. ago.	Catarrh.	r.	R. base.	Mod. 8 d. Grad.	12 d.	R.	0	—
494 W. Hamilton Allen, M.B.	F.	42	Temp. Suff.	Pneum.	1	2	Good. Good	1 son d. phth. 1 son 7	—	Pain in side.	r.	R. whole	Sev. 10 d.	14 d.	R.	0	—

496 Frank W. Cooper, L.R.C.S.	M.	8	Tot. abs. Suff.	Tonsill. Bron. Cat. Erysip.	1	2 1	Good. Ind.	M. chr. bron.	ago ; great suppuration Pertuss. } 1 y. Scarlat. } ago.	0	r.	R. base. 10 d. Grad.	10 d.	R.	0
497 G. H. Davis, L.R.C.P.	M.	24	Temp. Suff.	Pneum.	1		Good. Good.	F. d. phth. 56. 1 B. bronch.	—	0	r.	L. base. Mod. 7 d. Grad.	21 d.	R.	—
498 C. Harrison, M.D.	M.	10	Tot. abs. Suff.	Pneum.	2	4	Good. Good.	—	Weakly.	Cough, malaise.	0	Both bases. Sev. 14 d. Grad.	28 d.	R.	Recovered normal condition.
499 "	M.	14	Temp. Suff.	Pneum.	2	4	Good. Good.	0	—	Cough.	0	L. base. Sev. 5 d. Grad.	5 d.	R.	—
500 Jas. W. Harrison, M.R.C.S.	F.	30	Temp. Suff.	Pneum.		2	Bad. Ind.	F. d. phth. M. d. heart dis.	Colds.	0	r.	R. base. Sev. 14 d. Grad.	—	R.	0
501 Charles F. Hod- son, F.R.C.S.	—	54	Temp. Suff.	Pneum.	1		Good. Good.	1 son inelp. phth.	0	Catarrh, pains in back and limbs.	—	R. base, L. whole till D.	till D. 4th d.	D.	6
502 Edwin Jackson, M.R.C.S.	M.	15	Tot. abs. Suff.	Gastro- enterit. Tonsill. Bron. Cat. Pertussis.	1 1	2 4 3 2	Ind. Good.	M. pneum. F. chr. bron. S. strumous. F's S. prob. phth.	Measles 6 y. ago.	Headache 1 d.	r.	L. base. Sev. 9 d. Sud.	15 d.	R.	Parotitis.
503 "	M.	12	Tot. abs. Suff.	Tonsill. Bron. Cat. Erysip.	1	2 9 1	Good. Good.	F's S. phth. 1 B. chr. bron.	Hemopt. 4 m. ago.	Catarrh 3 d.	r.	R. base. L. whole Mod. 15 d. Grad.	never dis- appear- ing.		Softening under L. clavicle, phth.
504 "	M.	40	Intemp. Suff.	Scarlat. Measles. Febricula. Tonsill. Herpes. Bron. Cat. Erysip.	3 5 1 2 1 8 1	3 5 1 2 1 8 1	Good. Good.	0	Ae. rheum. 13 y. ago. Facial palsy 11 y. ago. Gonorrh.	Catarrh 6 d.	r.	L. whole Mod. till D.	D. 13th d.		

1. Labour and deliv. 36 h. aft. onset. 2. Husband pleuropneum. 2 m. later. 3. Convalescent; fell suddenly doud while dressing. 4. Pneum. came on in 3rd w of enteric fever. 5. 3 m. pregn.; labour 6th d.; mental anxiety for last 12 m.; D. from syncope or pericardial effusion. 6. Gangrene of lungs. 7.



Observer's Name.	Sex.	Age.	Temperate or other-wise. Food.	Concurrent cases of illness in	Same house.	Same district.	Sanitary state of house " " of district.	Family history of lung disease.	Previous illnesses of patient.	Premontory symptoms.	Mode of onset.	Part of lungs affected.	Character of fever. Duration Termination	Duration of physical signs.	Result.	Sequelæ.	Note.
505 Edwin Jackson, M.R.C.S.	F.	29	Temp. Suff.	Enteric. Ac. Rheum. Tonsill. Bron. Cat.	1	1	Ind. Good.	0	0	0	r.	L. base.	Mod. 8 d. Sud.	14 d.	R.	0	
506 Boyd Joll, M.B.	F	24	Temp. Suff.	0		2	Bad. Bad.	0	Inflammation of bowels.	0	r.	R. base.	Sev. 9 d. Grad.	12 d.	R.	0	
507 Duncan J. Mackenzie, M.D.	F.	4	Suff.	Tonsill.	1	1	Bad. Bad.	M's.F. } d.phth M's.B. }	0	Drowsiness, ob- irritab., pain in belly.	not ser- ved	Both bases.	Sev. till D.	till D.	D. sth d.		1
508 W. Mortimer, M.D.	M.	7	Temp. Suff.	Pneum. Ac. Rheum. Diphth. Pertuss. Catarrh. Tonsill. Bron. Cat. Erysip.	1	1 2 1 + 12 2 15 1	Ind. Ind.	0	Liable to <i>infl.</i> of <i>lungs</i> . Pertuss. 1 y. ago.	Catarrh 5 d.	r.	L. base.	Mod. 7 d. Grad.	30 d.	R.	0	2
509 Frederiek S. Palmer, M.D.	M.	50	Temp. Suff.	Catarrh.	1		Ind. Ind.	0	0	Succession of chills.	r.	R. apex.	Sev. 12 d. Sud.	16 d.	R.	Debility.	
510 Ed. B. Reckitt, L.R.C.P.	M.	25	Temp. Suff.	Pneum.	1		Ind. Good.	0	Enteric 6 y. ago.	0	r.	R. base.	Sev. 9 d. Grad.	14 d.	R.	0	
511 Amand Routh, M.D.	F.	25	Temp. Suff.	—			Good. Good.	0	0	Headache, pains in joints, each in side.	r.	L. whole	Sev. till D.	till D.	D. 7th d.		3
512 E. F. Scougal,	F.	39	Temp.				Ind.	0	Confined 2 m.	Pain in side.	0	L. base.	Mod.	7 d.	R.	Congestion	

		AGE.	SEX.	TEMP.	DIAGN.	IND.	1 B. pneum.	0	Cough, 1 w.	0	Both apices.	Sev. 10 d. Grad.	24 d.	R.	Debility.	
514	"	M.	6	Tot. abs. Suff.	Pneum. Bron. Cat.	2 4	Good. Ind.	1 B. pneum.	0	Cough 1 w.	0	Both apices.	Sev. 10 d. Grad.	24 d.	R.	Debility.
515	"	F.	3	Tot. abs. Suff.	—		Ind. Bad.	0	Cough, rigors, sleeplessness.	r.	L. base.	Sev. 11 d. Grad.	15 d.	R.	0	
516	"	F.	12	Tot. abs. Suff.	Scarlat. Diphth. Tonsill. Bron. Cat.	3 1 1	Good. Good.	0	Conval. from scarlat.	r.	R. whole L. base.	Sev. till D.	till D.	D. 24th d.		
517	W. B. Sellers, M.R.C.S.	M.	33	Temp. Suff.	—		Good. Good.	0	Ac. rheum. with heart dis. and congest. of lungs 9 y. ago.	—	Both bases.	Sev. 14 d. Grad.	—	R.	0	
518	W. D. Sheppard, L.R.C.P.	M.	26	Temp. Suff.	Pneum.	1	Ind. Ind.	0	Ac. bronch. 5 y. ago, R. pneum. 2 y. ago, R. pneum. 1 y. ago.	0	L. base.	Sev. 8 d. Sud.	21 d.	R.	0	
519	"	F.	32	Temp. Suff.	—		Ind. Ind.	0	Malaria 5 d.	—	L. base.	Mod. 8 d. Sud.	14 d.	R.	0	
520	Edwd. Skinner, L.R.C.P.	M.	29	Intemp. Suff.	Pneum. Scarlat.	2 4	Ind. Good.	M. bronch. 1 B. pneum.	0	Cough, pain.	—	R. base.	Sev. till D.	D. 8th d.		
521	"	F.	35	Temp. Suff.	Pneum. Scarlat.	2 4	Good. Good.	M. d. hæmopt. 29. F. d. hæmopt. 40. 1 B. nephrit.	—	—	r.	R. base.	Mod. 7 d. Grad.	11 d.	R.	0

1. D. by congestion of lungs. 2. Epistaxis 5th d. followed by fall of T. from 104° to 101°. 3. 3 m. pregn.; abortion just before D.; coma after that till D.; albuminuria. 4. Got wet sleeping out while drunk; D. by sudden dyspnoea; ? embolism.

Observer's Name.	Sex.	Age.	Temperate or otherwise. Food.	Concurrent cases of illness in	Same house.	Same district.	Sanitary state of house " " of district	Family history of lung disease.	Previous illnesses of patient.	Premontory symptoms.	Mode of onset.	Part of lungs affected.	Character of fever. Duration of fever.	Duration of physical signs.	Result.	Sequence.	Note.
522 Wm. White, M.D.	F.	66	Temp. Suff.	Pneum.	2	4	Good. Good.	0	0	Illness severe w. vomiting.	r.	Both bases.	Mild. 12 d. Grad.	28 d.	R.	—	1
523 "	F.	22	Temp. Suff.	Pneum.	2		Good. Good.	M. suf. from pneum. at same time in same house.	Delicate.	Headache, fever.	r.	R. base.	Sev. 10 d. Sud.	21 d.	R.	0	
524 "	F.	61	Temp. Suff.	Pneum.		4	Bad. Bad.	F. d. asth. and dropsy 50. M. d. bronch. 81.	Constant cough.	0	r.	L. base.	Mild. 7 d. Sud.	20 d.	R.	0	
525 M. M. Williams, L.R.C.P.	F.	27	Temp. Suff.	—			Ind. Ind.	—	0	—	r.	Both bases.	Sev. till D.	till D. 12th d.	D.		2
526 J. E. Adkins, M.R.C.S.	M.	46		0			Good. Good.	0	0	—	r.	Both bases.	Sev. till D.	till D. 11th d.	D.		
527 C. H. Cattle, M.D.	M.	29	Temp. Suff.	0			Ind. Good.	F.'s fam. phth.	0	Vomiting, scapular pain, head- ache.	r.	L. base	Mod. 8 d. Grad.	over 14 d.	R.	—	
528 T. Eytton-Jones, M.D.	M.	39	Temp. Suff.	Pneum. Measles. Catarrh. Bron. Cat.	1	+	Good. Good.	1 B. d. phth. 21.	0	0	r.	L. base.	Sev. 14 d. Grad.	16 d.	R.	—	0
529 "	M.	15	Tot. abs. Suff.	Measles. Diphth. Catarrh. Bron. Cat. Erysip.	+	+	Good. Good.	Great U. d. phth. 4 B.'s and 1 S. have weak chest.	—	Pain in chest.	—	R. whole	Sev. 14 d. Grad.	16 d.	R.	0	
530 "	M.	26	Temp.	Pneum.	1	+	Bad										

532	"	M.	19	Tot. abs. Suff.	Pneum.	1	Fair. Good.	0	Weak.	hand.	Cold.	r.	R. base.	Mod. 9 d. Sud.	14 d.	R.	0
533	J. Farrant Fry, L.R.C.P.	F.	74	Tot. abs. Suff.	Pneum. Catarrh. Tonsill. Bron. Cat.	+	Good. Good.	0	0	0	Malaise, dyspnoea.	—	R. base.	Mod. 8 d. Grad.	14 d.	R.	0
534	"	F.	39	Tot. abs. Suff.	0	0	Ind. Ind.	0	Pneum. 6 y. ago.		Shivering, pain in abd.	r.	R. apex. l. whole	Mild till D.	till D.	D. 10hd.	3
535	"	F.	64	Temp. Suff.	Pneum. Catarrh. Tonsill. Bron. Cat.	+	Good. Good.	0	Chr. bronch., gouty eczema.		Chill. malaise.	—	Both bases.	Mod. 5 d. Grad.	12 d.	R.	—
536	T. W. H. Gar- stang, M.R.C.S.	F.	5½	Tot. abs. Suff.	Scarlat. Tonsill.	1	Ind. Ind.	0	0		Vomiting.	0	R. whole	Sev. 14 d. Grad.	10 d. ?	R.	0
537	A. R. Graham, M.B.	F.	21	Temp. Suff.	Bron.	1	Good. Good.	F. bronch. M. d. phth. 28.	Cough occas.		0	r.	Both bases.	Mod. 12 d. Grad.	28 d.	R.	—
538	S. Griffith, M.D.	M.	66	Temp. Suff.	Pneum.	1	Ind. Ind.	0	0		0	r.	R. base.	Sev. 9 d. Sud.	21 d.	R.	—
539	James Holmes, M.D.	M.		Temp. Suff.	Pneum.	1	Good. Ind.	—	—		0	r.	R. whole	Sev. 11 d. Grad.	17 d.	R.	0
540	W. Jones-Morris, L.R.C.P.	M.	28	Temp. Suff.	0		Good. Good.	1 B. d. phth. 23.	0		0	r.	L. base.	Mod. 6 d. Grad.	21 d.	R.	0
541	F. W. Jordan, L.R.C.P.	F.	19	Tot. abs. Suff.	Diphth. Measles. Bron. Cat.	2	Ind. Ind.	—	Inf. of bowels.		0	r.	L. base.	Sev. 5 d. Sud.	5 d.	R.	0

1. 523 was daughter of 522; had been confined 5 w.; came to see M. and was attacked after being 1 w. in the house. 2. D. by asphyxia. 3. Symps. came on after long walk followed by heavy supper. 4. Peasoupy diarrh. from 2nd to 9th day; enteric suspected, but no spots, gurgling, or morning remissions of T. Had a drinking bout just before.



Observer's Name.	Sex.	Age.	Temperate or otherwise. Food.	Concurrent cases of illness in	Same house.	Same district.	Sanitary state of house " " of district	Family history of lung disease.	Previous illnesses of patient.	Premunitory symptoms.	Mode of onset.	Part of lungs affected.	Character of fever. Duration	Duration of physical signs.	Result.	Sequelæ.	Note.
542 Duncan J. Mackenzie, M.D.	M.	55	Temp. Insuff.	Ac. Rheum. Herpes. Bron. Cat.		1 1 1	Bad. Bad. Good. Good.	1 Daughter pleur. and 1 D. pneum. 5 m. ago.	0	Pain.	—	R. base.	Sev. 8 d. Grad.	8 d.	R.	Neuralgic pain at site of disease.	
543 L. W. Marshall, M.D.	M.	65	Temp. Suff.	Catarrh. Tonsill.		+	Good. Good.	0	Pneum. 20 y. ago.	Shivering,	r.	R. whole L. base.	Sev. 8 d. Grad.	25 d.	R.	—	
544 H. V. Palin, M.B.	F.	35	Temp. Suff.	Pneum. Bron. Cat. Erysip.	1	+	Ind. Good.	M. d. bronch. 1 S. d. plith.	0	—	r.	Both bases.	Sev. 15 d. Grad.	18 d.	R.	0	
545 D. M. Williams, L.K.Q.C.P.	M.	45	Temp. Suff.	Bron. Cat.		1	Ind. Ind.	0	Pleurisy 10 y. ago.	Purging, vomiting.	r.	R. base.	Sev. 11 d. Grad.	21 d.	R.	0	
546 " "	F.	42	Temp. Suff.	0			Ind. Ind.	0	Bronch. oft.	0	r.	L. base.	Mild. 20 d. Grad.	25 d.	R.	0	
547 " "	F.	40	Temp. Suff.	Tonsill. Bron. Cat.		1 2	Ind. Ind.	Several d. plith.	Catarrh oft.	0	r.	R. base.	Mod. 10 d. Grad.	30 d.	R.	0	
548 " "	M.	8½	Tot. abs. Suff.	0			Good. Good.	M. bronch. oft.	Bronch. thrice Pneum. 5 y. ago.	Catarrh, bronch.	?	R. base.	Sev. 10 d. Grad.	14 d.	R.	0	
549 " "	M.	50	Tot. abs. Suff.	0			Good. Good.	0	0	0	r.	L. base.	Sev. till D.	till D.	D.	—	1
550 Edw. Williams, M.D. (Wrexham).	M.	67	Temp. Suff.	Pneum.	1		Good. Good.	0	0	Sudden attack after exposure on a mountain side on a very cold	—	R. base.	Sev. 8 d. Grad.	27 d.	R.	—	

M.	21	Temp. Sufl.				Ind.	4 Ba & Ss d. pht.	ago.				5 d. Grad.				
551 E. Williams, L.R.C.P. (Bala).																
552 Owen Williams, L.K.Q.C.P. (Rhosygaer).	M.	21	Tot. abs. Sufl.	Pneum.	1	Good. Good.	4 Ba & Ss d. pht.	0	Cough, head- ache, do- lirium, pain in chest.	r.	Both bases.	Sev. 11 d. Grad.	14 d.	R.	0	
553 "	M.	36	Intemp. Sufl.	Pneum. Tonsill. Erysip. Pertuss.	1 5 3 +	Ind. Ind.	0	0	Vomiting.	r.	R. whole	Mod. 7 d. Grad.	26 d.	R.	—	
554 "	M.	40	Temp. Insuff.	Pneum. Tonsill. Herpes. Erysip	1 3 3 1	Ind. Ind.	"Something" matter with chest 10 y. ago.	"Chest affect ed" 10 y. ago.	—	r.	Both bases.	Sev. 8 d. Sud.	30 d.	R.	0	
555 Wm. Berry, L.R.C.P.	M.	35	Temp. Insuff.	—		Bad. Bad.	—	Bronch. 1 y. ago.	Malaise and chills 3 d.	0	L. base.	Mild. 7 d. Grad.	15 d.	R.	0	
556 M. G. Biggs, M.R.C.S.	F.	32	Temp. Sufl.	Pneum. (Cat.) Measles. Catarrh. Tonsill. Bron. Cat. Erysip. Diarrhoea.	2 6 3 1 2 1 +	Ind. Ind.	0	Syph. lately. Delicate and hysterical.	Malaise, pain in chest.	0	L. base.	Mod. 6 d. Sud.	19 d.	R.	Cough, with pain on left side.	
557 G. F. Blake, M.R.C.S.	F.	6	Tot. abs. Sufl.	Pneum. Catarrh. Herpes. Bron. Cat.	1 3 1 1	Ind. Good.	M. chron. bronch.	Measles 3 y. ago.	0	r.	L. base.	Mod. 2 d. Sud.	5 d.	R.	0	
558 E. O. Daly, M.B.	M.	12	Tot. abs. Sufl.	Pneum.	1 in same ship.	Good.	0	Abscess over L. trochan- ter 3 m. ago.	Pain in chest, headache.	r.	R. apex.	Mod. 3 d. Sud.	8 d.	R.	0	
559 T. W. H. Gar- stang, M.R.C.S.	M.	19	Temp. Sufl.	Pneum. Tonsill. Bron. Cat.	1 1 1	Good. Ind.	0	0	Vomiting.	—	R. base.	Mod. 8 d. Grad.	till D. 10th d.	D.		2
560 "	M.	22	Temp. Sufl.	Pneum. Tonsill. Bron. Cat.	3 1 1	Good. Ind.	0	0	Malaise, pain in side.	0	L. base.	Sev. 12 d. Grad.	21 d.	R.	0	

1. Overwork. 2. Overworked; T. normal on 9th d.; sudden D. on 10th d. without warning or possibility of aid; no P.M.

Observer's Name.	Sex.	Ago.	Temperate or other-wise. Food.	Concurrent cases of illness in	Same house.	Same district.	Sanitary state of house " " of district	Family history of lung disease.	Previous illnesses of patient.	Premontory symptoms.	Mode of onset.	Part of lungs affected.	Character of fever. Duration Termination	Duration of physical signs.	Result.	Sequelae.	Note.
561 Frede. H. Haynes, M.D.	F.	47	Temp. Suff.	Enteric. Catarrh. Tonsill. Herpes. Bron. Cat.	1	few ++++	Good. Good.	1 S. asth., d. phth. 1 B. subj. to ind. of lung.	0	Headache, dyspnoea, pains, cold.	r.	R. base.	Mod. 10 d. Sud.	18 d.	R.	—	
562 E. H. Howlett, F.R.C.S.	F.	8	Suff.	—		++	Ind. Ind.	0	—	0	r.	L. base.	Mod. 8 d. Sud.	14 d.	R.	0	
563 H. W. Laing, M.D.	M.	20	Temp. Suff.	Pneum.	1	0	Good. Good.	0	Infl. of lungs 11 y. ago.	0	r.	R. base.	Mod. 10 d. Grad.	14 d.	R.	0	1
564 J. Roche Lynch, L.R.C.P.	M.	35	Temp. Suff.	Pneum.	1		Good. Ind.		Plour. some y. ago; intermittent fever, spleen hard and big.	Pain in side, restlessness, fever, foul tongue, anxiety, depression.	r.	R. base.	Sev. 3 d. Grad.	Over 14 d.	R.	0	2
565 J. A. Mackenzie, M.B.	F.	25	Temp. Suff.	Pneum. Scarlat. Enteric. Bron. Cat.	1	2 1 1 2	Ind. Ind.	F. d. pneum. 4l.	Erysip. of head and face, "never well since."	Pain in side, sickness.	r.	Both bases.	Sev. 5 d. Grad.	8 d.	R.	Phlebitis L. leg 10th d., R. leg 13th d.	3
566 James Mackinlay, L.R.C.P.	M.	20	Temp. Suff.	Enteric.		1	Good. Good.	M.'s M. d. phth.	0	Bron. cat. 3 w.	—	L. base.	Sev. 7 d. Grad.	27 d.	R.	0	4
567 F. B. Mallet, M.D.	M.	59	Temp. Suff.	0			Good. Good.	0	0	Pain, shivering.	—	Nearly whole of both from bases.	Mod. till D.	till D. 8th d.	D.		
568 John M. H. Martin, M.D.	F.	55	Temp. Suff.	Pneum.		1	Good.	0	Always	Vomiting.	r.	R. base.	Sev.		D.		

	Suit.	1	2	3	4	Ind.	chest.	ago.	sickness, vomiting, chills.		L. base.	9 d. Grad.	21 d.	R.	—
570 J. Matthews, L.R.C.P.	Temp. Suff.	12	Pneum. Tonsill. Herpes. Bron. Cat.	1	1	Good. Good.	0	Pertuss. last y. cat.	Cough.	0	L. base.	Mod. 14 d. Grad.	21 d.	R.	—
571 T. W. Moorhead, M.D.	Temp. Suff.	55	Pneum.	1	1	Ind. Ind.	0	Dyspepsia.	0	r.	R. apex.	Mod. 14 d. Grad.	20 d.	R.	0
572 A. Raynor, M.D.	Tot. abs. Suff.	5	Pneum. Catarrh. Bron. Cat. Erysip.	4 + + 1	4	Good. Good.	0	0	0	r.	R. apex.	Mod. 9 d. Sud.	15 d.	R.	0
573 H. G. Plimmer.	Temp. Suff.	7	Measles. Herpes. Bron. Cat.	10 1 5	10	Ind. Ind.	0	Tonsill 5 y. ago, measles this y.	Fever, cough.	0	Both bases.	Mod. 9 d. Sud.	16 d.	R.	0
574 T. H. Moorhead, M.D.	Temp. Suff.	20	Pneum.	2	2	Ind. Ind.	Phth. on both sides.	—	Cough.	r.	Both bases.	Mod. 25 d. Grad.	30 d.	R.	—
575 T. F. Raven, L.R.C.P.	Temp. Suff.	6	Pneum.	3	3	Bad. Ind.	Phth. on both sides.	Scarlat. 2 y. ago. Enteric } last Measles } y.	Catarrh.	?	R. apex.	Mod. 6 d. Sud.	12 d.	R.	Cough, debility.
576 "	Temp. Suff.	5	Pneum. Rotheln.	3 2	3	" "	" "	Scarlat. 2 y. Measles last y.	Cough. Toothache.	r.	L. base to apex.	Sev. 8 d. Sud.	14 d.	R.	Cough, debility.
577 "	Temp. Suff.	9	Pneum. Rotheln. Scarlat.	3 1 1	3	" " "	" " "	Scarlat. 2 y. ago ; infant remit. fever 6 y. ago ; measles 7 y. ago.	Catarrhal fev. 2 w. Cough.	r.	Both apices.	Sev. 9 d. Sud.	16 d.	R.	Debility.
578 "	Tot. abs. Suff.	2	Pneum. Scarlat.	3 1	3	" "	" "	Measles last y.	Cough.	?	L. base to apex.	Mod. 16 d. Grad.	22 d.	R.	Cough, debility, anemia.
579 "	Temp. Suff.	4	Pneum. Scarlat.	4 1	4	" "	" "	Measles last y.	Sickness. Diarrhoea.	r.	0	Mod. 3 d. Sud.	0	R.	—

1. 4th d. sudden syncope and threatened collapse. 2. Seventy grains of quinine taken on rigor; slight dulness was only effect; has lived on West African coast.  
3. Onset 14 d. after labour; breech case; midwife; child stillborn. 4. Exp. sun all day; chill in evening. 5. All the cases occurred in one house; 576 onset 15 d. after 575; 577 onset 3 d. after 576; 578 onset 5 d. after 577; 579 onset 2 d. after 578; no enteric symps. 6. Nothing but supposed septic influence to account for this case; ? abortive pneumonia.



Observer's Name.	Sex.	Age.	Temperate or otherwise. Food.	Concurrent cases of illness in	Same house.	Same district.	Sanitary state of house " " of district	Family history of lung disease.	Previous illnesses of patient.	Premunitory symptoms.	Mode of onset.	Part of lungs affected.	Character of fever. Duration Termination	Duration of physical signs.	Result.	Sequelæ.	Note.
580 T. F. Raven, L.R.C.P.	M.	37	Intemp. Suff.	Pneum. Scarlat.		3 1	Ind. Good.	0	Enteric last y. Catarrh.	0	r.	L. apex.	Mod. till D.	till D.	D. 6th d.		1
581 "	M.	57	Intemp. Insuff.	Pneum. Scarlat.		3 1	Good. Good.	0	0	0	r.	L. base.	Mod. till D.	till D.	D. 13thd.		2
582 "	M.	53	Temp. Suff.	Pneum. Enteric.		3 1	Good. Good.	0	0	Bron. Cat. 2 w.	r.	R. base to apex.	Mod. 8 d. Sud.	18 d.	R.	—	3
583 "	F.	39	Temp. Suff.	Pneum. Enteric.		3 1	Good. Good.	0	Chr. bronch. Emphysema.	Pain in side.	r.	L. base.	Mild 7 d. Sud.	till D.	D. 12thd.		4
584 George Shearer, M.D.	M.	2	Tot. abs. Suff.	Pneum. Fever. Catarrh. Bron. Cat.	1	3 4 3 2	Good. Good.	0	0	Chills.	r.	L. whole	Sev. 4 d. Sud.	11 d.	R.	Dull per- cussion completely cleared up on 11th d.	
585 "	F.	5	Tot. abs. Suff.	Pneum. Scarlat. Typhus. Enteric. Catarrh. Tonsill. Bron. Cat.		3 1 1 1 3 1 2	Good. Good.	0	0	0	r.	—	Sev. 4 d. Sud.	—	R.	0	
586 "	M.	23	Temp. Suff.	Ditto.		"	Bad. Good.	0	0	0	r.	R. base.	Sev. 4 d. Sud.	12 d.	R.	—	
587 H. Conpland Taylor, M.D.	F.	23	Temp. Suff.	Pneum.	1		Bad. Ind.	0	0	Sore throat; feeling ill some time.	0	Both bases.	Sev. 35 d. Grad.	35 d.	R.	—	5
588 T. W. Thursfield, M.D.	F.	7	Tot. abs. Suff.	Catarrh.		+	Good. Good.	M's M's fam. delicate; 2 of them d. within	Portuss. 6 m. ago. Capill. bron.	Cough, fever.		Both bases.	Sev. 6 d. Sud.	10 d.	R.	Dobility, anæmia.	

590 S. Warren, M.D.	M.	21	Tot. abs. Suff.	Pneum. Bron. Cat.	3 +	Good. Good.	0	0	0	r.	Both whole.	Grad. Sev. 35 d. Grad.	R.	0	6
591 Vere G. Webb, L.K.Q.C.P.	M.	37	Intemp. Suff.	Tonsill. Herpes. Bron. Cat Erysip.	1 5 2 3	Ind. Good.	0	Erythema, bilious attacks.	0	r.	Both bases.	Mod. till D.	D. 10th d.		7
592 Thomas F. Young, L.K.Q.C.P.	M.	25	Temp. Suff.	Enteric.	2	Ind. Ind.	F. d. bronch. 1 S. d. pneum.	Brouch. 9 m. ago.	0	r.	R. whole	Sev. till D.	D. 11th d.		8
593 J. M. H. Martin, M.D.	F.	29	Temp. Suff.	—		Good. Ind.	2 S. and 1 B. delicate chests.	Delicate.	Sickness, chills, pains.	r.	L. whole	Sev. till D.	—	D.	9
594 T. H. Moorhead, M.D.	M.	65	Temp. Suff.	Pneum.	1	Ind. Ind.	0	0	Cough.	r.	R. base.	Mod. 12 d. Sud.	R.	0	
595 E. T. Wilson, M.B.	M.	74	Temp. Suff.	Pneum. Tonsill. Herpes. Bron. Cat.	1 + + +	Good. Good.	0	Sciatica, lithic acid deposits.	Pain in thighs.	r.	Both bases.	Mod. till D.	D. 7th d.		10
596 Geo. G. Whitwell, M.B.	F.	15	Tot. abs. Suff.	Bron. Cat.	1	Ind. Good.	M.'s B. d. phth.	0	Headache } Sickness }	0	R. base.	Mod. 6 d. Grad.	R.	0	11
597 J. A. Erskine Stuart, L.R.C.S.	F.	54	Temp. Suff.	Pneum. Enteric. Catarrh. Tonsill. Bron. Cat. Erysip. Diphth.	7 5 2 1 8 1 3	Bad. Ind.	0	Brouch.	Headache.	0	Both bases.	Mod. 7 d. Sud.	R.	0	12
598 "	M.	19	— Suff.	Enteric. Tonsill.	2 1	Ind. Ind.	F. } 2 F.'s S. } d. } phth }	0	Headache, anorexia.	0	R. whole	Mild. 7 d. Grad.	R.	—	
599 E. S. Scott, M.B.	M.	35	Tot. abs. Suff.	Tonsill.	1	Ind. Good.	0	0	0	r.	R. whole L. apex.	Sev. till D.	D. 9th d.		

1. Diarrhoea, albuminur, jaundice. 2. P. M. Grey hepatiz. L. lower lobe. 3. Transient albumin. 4. Nursing a case of pneum. just before; P. M. L. lower lobe purulent at base, red above; rest of lungs oedema and emphysema. 5. 1 S. d. in same house of pneum. on 6th d.; ill at same time; foul sink con. with drains; 587 had ulcerated throat, brown, swollen tongue, delirium, and jactitation; no spots, tympanites, or tenderness of abdomen. 6. R. lung first affected in whole extent, when resolution had begun, L. lung attacked; when L. lung clearing, R. base attacked again. 7. Temp. lately; colliquative diarrh. on 12th d., fatal in 24 h. 8. D. by dyspnoea. 9. D. by exhaustion. 10. D. by coma. 11. Privy emptied; complained of bad smells during process; onset next d.; well before. 12. Escape of sewer gas from sink-pipe.

Observer's Name.	Sex.	Age.	Temperate or otherwise. Food.	Concurrent cases of illness in	Same house.	Same district.	" " of district.	Family history of lung disease.	Previous illnesses of patient.	Prodromitory symptoms.	Mode of onset.	Part of lungs affected.	Character of fever. { Duration Termination } of fever.	Duration of physical signs.	Result.	Sequelæ.	Note.
600 E. S. Scott, M.B.	F.	35	Temp. Suff.	Tonsill. Herpes. Bron. Cat. Scarlat.		7 1 1 4 4 +	Good. Good.	F. and F.'s F. asthma.	Asthma.	0	r.	R. base.	Sev. 8 d. Grad.	15 d.	R.	0	
601 "	F.	35	Temp. Suff.	Pneum. Bron. Cat.		2 1	Good. Good.	M. } d. pneum. S. } F. bronch.	Confined 16 d. ago.	0	r.	R. base.	Sev. till D.	till D.	D. 10th d.		
602 "	F.	46	Temp. Suff.	Tonsill. Bron. Cat. Erysip.		1 3 1	Ind. Good.	M. d. phth.	Pleur. } 5 y. } Pneum. } ago.	Indigestion.	0	R. whole	Sev. till D.	till D.	D. 10th d.		
603 A. C. Rayner, M.D.	F.	71	Temp. Suff.	Enteric. Scarlat.		3 6	Good. Good.	0	Bronch.	Vomiting, purging.	0	R. base.	Mod. till D.	till D.	D. 6th d.		
604 J. J. Palmer, M.R.C.S.	M.	43	Temp. Suff.	—			Ind. Ind.	0	Ac. rheum.	Chilliness, catarrh.	r.	L. base.	Mod. 8 d. Sud.	Over 30 d.	R.	Indifferent expansion.	1
605 S. H. Owen, M.D.	F.	5	Tot. abs. Suff.	Tonsill. Bron. Cat.		1 5	Ind. Good.	M.'s 2S. } d. M.'s B. } phth 1 B. d. bron.	Convulsions, bronch.	0	r.	L. base.	Sev. 7 d. Sud.	12 d.	R.	0	
606 T. William Newman, M.D.	M.	49	Temp. Suff.	0			Good. Ind.	0	Hæmaturia, bronch., D. T. once.	Malaise, prostration.	r.	R. base.	Sev. 10 d. Grad.	15 d.	R.	0	2
607 Thomas Morgan, L.K.Q.C.P.	M.	—	Temp. Suff.	Tonsill. Herpes.	1 1		Ind. Good.	0	<i>Infl. of lungs.</i>	0	r.	L. base.	Sev. 11 d. Grad.	16 d.	R.	0	
608 W. A. Michio, M.B.	M.	10	Temp. Suff.	Pneum.	1	2	Ind. Ind.	0	0	0	r.	L. base.	Sev. 5 d. Sud.	14 d.	R.	—	
609 "	M.	47	Temp. Suff.	Pneum. Enteric.	1	2 3	Ind. Ind.	0	0	0	r.	R. apex.	Mod. 4 d. Sud.	till D.	D. 10th d.		3





Observer's Name.	Sex.	Age.	Temperate or otherwise. Food.	Concurrent cases of illness in	Same house.	Same district.	Sanitary state of house " " of district	Family history of lung disease.	Previous illnesses of patient.	Premontory symptoms.	Mode of onset.	Part of lungs affected.	Character of fever. Duration Termination	Duration of physical signs.	Result.	Sequence.	Note.
619 W. T. Cant, L.R.C.P.	F.	17	Tot. abs. Suff.	Pneum. Scarlat. Tonsill. Herpes. Bron. Cat. Erysip.		4 3 2 1 3 1	Good. Good.	1 S. congestion of lungs.	Anemia.	Faintness, vomiting.	r.	R. base.	Sev. 11 d. Sud.	42 d.	R.	0	
620 Macfie Campbell, M.D.	M.	6	Suff.	Scarlat. Measles. Varicella. Catarrh. Bron. Cat. Erysip.		2 1 1 2 1 1	Good. Good.	0	Bilious attack with fever 2 y. ago, measles 3 m. ago.	Bilious vomiting 1 d.	0	R. base.	Sev. 8 d. Grad.	14 d.	R.	0	
621 Charles Broomhead, M.D.	M.	13	Tot. abs. Suff.	Tonsill. Bron. Cat.		2 3	Bad. Good.	U. d. phth.	Measles. Scarlat.	Anorexia, chilliness.	r.	R. whole	Sev. 8 d. Sud.	15 d.	R.	0	
622 Fredk. G. H. Daly, M.D.	F.	30	Temp. Suff.	Pneum.	5		Good. Good.	0	—	0	r.	—	Sev. till D.	till D.	D.		1
623 James Brown, M.B.	M.	22	Temp. Insuff.	Pneum.		3	Bad. Bad.	F. d. pneum.	0	—	r.	R. whole L. base.	Sev. 12 d. —	20 d.	R.	—	
624 Charles Boyce, M.B.	F.	27	Temp. Suff.	Bron. Cat. Erysip.		1 1	Good. Good.	0	0	Malaise 14 d., pains.	—	L. base.	Sev. 7 d. Sud.	13 d.	R.	0	
625 "	M.	41	Temp. Suff.	Scarlat. Tonsill. Erysip.		1 2 1	Good. Good.	F. d. phth. 49.	0	Sickness.	r.	R. base.	Sev. 17 d. Grad.	20 d.	R.	—	
626 James Allan, M.D.	M.	17	Intermp. Insuff.	—			— —	—	—	Headache, anorexia, pain in chest.	0	R. apex.	Sev. 7 d. Sud.	13 d.	R.	0	2
627 "	M.	38	Intermp. Suff.	—			— —	—	—	—	—	Both bases.	Mod. till D.	till D.	D. 3rd d.		3



Observer's Name.	Sex.	Age.	Temperate or other-wise. Food.	Concurrent cases of illness in	Same house.	Same district.	Sanitary state of house " " of district	Family history of lung disease.	Previous illnesses of patient.	Premontory symptoms.	Mode of onset.	Part of lungs affected.	Character Duration Termination of fever.	Duration of physical signs.	Result.	Sequelæ.	Note.
639 G. A. Phillips, M.R.C.S.	M.	6	Tot. abs. Suff.	Pneum. Herpes.	3 1		Bad. Good.	0	Weakly, rickety.	Fever.	r.	Both bases.	Sev. 5 d. Grad.	14 d.	R.	0	1
640 "	M.	14	Tot. abs. Suff.	"	"		" "	0	0	Fever, delirium.	r.	Both bases.	Sev. 10 d. Sud.	14 d.	R.	0	
641 J. Quirke, M.R.C.P.E.	M.	8	Tot. abs. Suff.	Pneum. Catarrh. Tonsill. Herpes. Bron. Cat.	1	4 1 1 6	Good. Good.	M. d. phth. 30.]	0	Malaise, pain in side.	r.	L. base.	Sev. 10 d. Grad.	12 d.	R.	0	
642 W. M. A. Wright, M.B.	M.	25	Tot. abs. Suff.	Pneum.	1	+	Ind. Good.	0	0	Malaise 1 d.	r.	R. base.	Mod. till D.	till D.	D. 19th d	—	2
643 Walter G. Smith, M.D.	F.	82	Temp. Suff.	Pneum.	1		Good.	—	—	Vomiting, pain in side, cough, prostration.	—	R. apex.	Mod. Grad.	—	D. 10th d	—	
644 Henry G. Terry, M.R.C.S.	M.	9 12	Tot. abs. Suff.	Pneum. Enteric. Tonsill. Bron. Cat.		1 1 1 1	Good. Ind.	0	Bron. 6 m. ago.	Vomiting.	0	L. base.	Mod. till D.	till D.	D. 10th d		3
645 D. H. Forty, L.R.C.P.	M.	56	Temp. Suff.	" 0			Good. Good.	—	—	0	r.	R. whole L. base.	Mod. till D.	till D.	D. 7th d.		
646 J. Farrar, L.R.C.P.	F.	58	Temp. Suff.	Enteric. Catarrh. Tonsill. Bron. Cat. Erysip.		some some 1	Good. Ind.	0	Bronch., ear- bunches last 2 y. causing weakness.	Pain in side, cough, wiry quick pulse, face pinched.	r.	R. base.	Mod. 7 d. Grad.	14 d.	R.	0	4
647 J. T. W. Lips- comb, M.D.	M.	43	Temp. Suff.	" 0			Ind. Ind.	F. d. chr. bronch. 63.	0	0	r.	R. base.	Sev. 9 d. Grad.	12 d.	R.	0	
648 A. Dunbar Walker, M.D.	F.	27	Temp. Suff.	Scarlat. Tonsill.		3 1	Good. Good.	0	—	0	r.	L. base.	Mod. 8 d. Grad.	12 d.	R.	0	

649 O. Bowen, M.R.C.S.	M	21	Temp. Suff.	—	0	?	Good. Good.	0	0	0	0	r.	R. base.	Mod. 8 d. Grad.	7 d.	R.	0
650 David Turnbull Smith, M.B.	F.	60	Tot. abs. Suff.	—			Good. Good.	0	Pleurisy.	Pleuritic pain, bilious vomiting.	r.	R. base.	Sev. 7 d. Grad.	14 d.	R.	—	
651 Wm. White, M.D.	F.	57	Temp. Suff.	Pneum. Enteric. Bron. Cat.	3 2 +	3 2 +	Good. Ind.	0	0	Out of sorts 1 w.	r.	R. base.	Mod. 5 d. Sud.	10 d.	R.	—	
652 "	M.	62	Fairly temp. Suff.	Enteric. Tonsill.	few few	few few	Good. Ind.	F. and B's. chest diseases.	Congestion of lungs, bron. often lately.	0	r.	R. base.	Mod. 5 d. Sud.	21 d.	R.	—	
653 C. Broomhead, M.D.	F.	11	Tot. abs. Suff.	Enteric. Tonsill. Horpes. Bron. Cat.	2 1 3 1 4	2 1 3 1 4	Bad. Ind.	F. pneum.	—	0	r.	L. base.	Sev. 5 d. Sud.	10 d.	R.	0	
654 John Reid, M.B.	M.	18	Tot. abs. Suff.	Scarlat. Bron. Cat. Erysip.	3 + 2	3 + 2	Good. Good.	—	Scrofulous.	Malaise, chilliness.	r.	R. base.	Sev. 6 d. Grad.	18 d.	R.	Endo- carditis.	
655 G. T. Schofield, L.R.C.P.E.	M.	20	Tot. abs. Suff.	Pneum.	1	1	Good.	0	0	Pain in chest	r.	R. whole	Sev. 8 d. Grad.	13 d.	R.	0	
656 "	M.	22	Temp. Suff.	Pneum.	1	1	Good. Good.	0	Pneum. as a child.	Pain in side.	r.	R. whole	Sev. 10 d. Grad.	14 d.	R.	0	
657 Herbert S. Renshaw, M.D.	M.	31	Intemp. Suff.	Pneum. Bron. Cat.	2 1	2 1	Ind. Good.	F. bronch. 1 B. } d. phth. 1 S. } 1 B. pneum.	Pneum. 7 y. ago.	0	r.	L. whole	Sev. 7 d. Sud.	16 d.	R.	0	
658 John Brown, L.R.C.P.	F.	21	Tot. abs. Suff.	Pneum.	1	1	Good. Good.	1 B. d. phth.	0	—	r.	R. base.	Mod. 3 d. Grad.	3 d.	R.	Slight cough.	
659 "	M.	3	Tot. abs. Suff.	Pneum. Bron. Cat.	1 2	1 2	Good. Good.	0	—	—	—	Both bases.	Sev. 6 d. Grad.	11 d.	R.	0	

1. Three children attacked in same house; first child not under my care; 639 attacked 14 d. later; 640 14 d. after 639; house stands by itself; no proper water supply; clay subsoil. 2. D. by dyspnoea. 3. Sudden onset while crossing Bristol Channel. 4. Chill. 5. Relapse on 15th d.; T. rose for 1 d.; rusty sputa for 6 d.



Observer's Name.	Sex.	Age.	Temperato or other- wise. Food.	Concurrent cases of illness in	Same house.	Same district.	Sanitary state of house " " of district	Family history of lung disease.	Previous illnesses of patient.	Premontory symptoms.	Mode of onset.	Part of lungs affected.	Character Duration Termination of fever.	Duration of physical signs.	Result.	Bequest.	Note.
660 John Brown, L.R.C.P.	M.	21	Tot. abs. Suff.	Pneu. (Cat. Mumps. Catarrh. Tonsill. Herpes. Bron. Cat. Erysip.		4 + 2 6 1 13 1	Bad. Good.	0	0	Fever.	r.	R. base.	Mod. 3 d. Sud.	3 d.	R.	0	
661 S. Nesfield, M.D.	M.	60	Intemp. Suff.	—			Ind. Ind.	0	0	Fever, pain in side and joints.	0	R. base.	Mod.	—	D. 46th d	Chronic ab- scess and gangrene R. lung.	
662 S. D. Clipping- dale, M.D.	M.	40	Temp. Suff.	—			Good. Good.	—	—	Lassitude, anorexia.	r.	R. base.	Sev. 7 d. Sud.	Still.	R. part.	—	1
663 "	M.	40	Temp. Insuff.	—			Ind. Ind.	—	—	—	r.	R. base.	Mod.	31 d.	R.	—	
664 "	M.	13	Tot. abs. Suff.	—			Ind. Good.	M. chr. bron.	—	—	—	R. base.	Sev. 5 d. Sud.	See Note.	R.	0	2
665 "	M.	11	Tot. abs. Suff.	Bron. Cat.	3	+	Good. Good.	F. d. phth.	Pertussis in infancy.	—	—	L. base.	Sev. 5 d. Sud.	5 d.	R.	—	
666 "	M.	7	Tot. abs. Insuff.	—			Ind. Good.	M. chr. bron. and emphys. B. d. "congest. of lungs."	—	—	—	L. base.	Mod. till D.	till D.	D. 4th d		
667 S. H. Whitaker, L.R.C.P.	M	65	Intemp. Suff.	0	1		Ind. Ind.	0	Dyspepsia.	Anorexia, pain in side, sleeplessness.	r.	R. base.	Mod. till D.	till D.	D. 8th d		
668 J. J. Marshall, L.R.C.S.I.	M.	53	Temp. Suff.	Pneum. Scarlat. Catarrh. Tonsill. Bron. Cat.	1	1 2 1 2	Ind. Good.	M. d. bronch. 79.	Pneum. twice, rheumat. occas.	0	r.	R. base.	Mod. 4 d. Grad.	14 d.	R.	0	

669 J. Jenkin Lloyd, L.R.C.P.	M.	32	Intemp. Suff.	Pneum. Tonsill. Erysip. Enteric. Scarlat.	0 + 5 2 +	0 Bad. Bad.	0 0 0	pneum. last y.	Pain in side, diarrhoea.	0	R. base.	5 d. Grad.	3
670 "	M.	30	"	"	"	Ind. Bad.	0	0				14 d.	—
671 D. Arthur Davies, M.D.	M.	68	Temp. Suff.	Measles. Bron. Cat.	3 2	Good. Good.	0	<i>Ind. R. lung</i> 30 y. ago.	0	r.	R. whole.	Mod. 12 d. Grad.	Excessive sweating for many days.
672 G. H. Batter- bury, M.D.	F.	64	Temp. Suff.	—		Ind. Ind.	F. d. phth.	Epilepsy, fibroid of uterus, bronch. 2 y. ago.	Cough and catarrh 1 w.	r.	R. base.	Mod. 8 d. Sud.	There is still loose crepita- tion at R. base.
673 Wm. White, M.D.	M.	12	Temp. Suff.	Pneum. Fever. Tonsill. Bron. Cat.	3 1 2 +	Bad. Ind.	0	Winter cough.	0	r.	L. base.	Mod. 12 d. Grad.	0
674 B. Addy, M.D.	M.	4½	Tot. abs. Suff.	Pneum. Tonsill. Bron. Cat.	1 1 +	Good. Good.	U. d. phth.	Measles 3 y. ago.	Cerebral symps., fever.	0	L. whole	Sev. 7 d. Sud.	0
675 R. R. Lloyd, M.R.C.S.	M.	47	Intemp. Suff.	0		Good. Good.	0	Gastritis twice, hepatitis, low fever.	0	r.	L. whole	Sev. 7 d. Sud.	—
676 B. H. Munnby, M.D.	M.	32	Tot. abs. Suff.	Herpes.	1	Ind. —	1 U. } d. phth. 1 A. }	Ac. rheum. 4 y. ago.	Malaise 1 d.	r.	R. base.	Sev. 13 d. Grad.	Anæmia, weakness.
677 J. Mulvany, M.D.	—	7	Temp. Suff.	Bron. Cat.	1	Good. Ind.	0	—	Bronch. 3 d., headache, somnia- lence, vomiting.	—	Both bases.	Sev. 10 d. Grad.	0
678 A. O. Grosvenor, M.D.	M.	28	Temp. Suff.	—		Good. Good.	0	0	Pleuritic pains.	0	Both bases.	Mod. 7 d. Grad.	0

1. 5 m. later the physical signs are normal. 2. Attack was followed by a condition resembling emphysema; great incr. of reson., expirat. prolonged, and dyspnoea; this lasted 14 d., and gradually subsided, leaving no abnormal signs. 3. Sympts. at first resembled enteric. 4. Relapse on 11th d. lasting 2 d.

Observers	Sex.	Age.	Temperate or other- wise. Food.	Concurrent cases of illness in	Same house.	Same district.	Sanitary state of house " " of district	Family history of lung disease.	Previous illnesses of patient.	Premunitory symptoms.	Mode of onset.	Part of lungs affected.	Character Duration Termination of fever	Duration of physical signs.	Result.	Sequelæ.	Note.
679 L. Druitt, M.D.	M.	1½	— Insuff.	Pneum. Measles. Tonsill. Bron. Cat. Erysip.	3	+	Bad. Bad.	F. plith.	Measles same m.	Catarrh.	?	Both bases.	Sev. till D.	till D.	D. 16th d		
680 D. O. Fountaine, L.R.C.P.	M.	25	Temp. Suff.	Pneum.	1	1	Good. Good.	0	Jamnicol m. ago.	Pain, nausea, fever, cough.	r.	R. whole	Sev. till D.	till D.	D. 7th d.		
681 R. W. Gentles, L.R.C.P.E.	M.	27	Tot.abs. Suff.	Pneum. Scarlat. Tonsill. Bron. Cat.	1	2 1 2 3	Ind. Ind.	0	0	0	r.	R. base.	Mod. 6 d. Grad.	7 d.	R.	Ocasional slight hemopt.	
682 M. T. Yarr.	F.	38	Temp. Suff.	Pneum. Measles. Tonsill. Bron. Cat.	1	1 0 3	Good. Good.	0	0	0	r.	R. base.	Mild 7 d. Sud.	9 d.	R.	0	
683 D. W. Currie, M.B.	F.	6	Tot.abs. Suff.	Pneum. Measles. Tonsill. Bron Cat.	1 2	2 2	Good. Ind.	M. bron. cat.	Measles last y., ac. bron. 2 y. ago.	0	r.	R. base	Sev. 8 d. Sud.	21 d.	R.	0	
684 "	M.	52	Temp. Suff.	Pneum. Tonsill. Bron. Cat. Erysip. Scarlat.	2 6 2 1 5	+	Ind. Ind.	M. d. chr. bron. F. pneum. S. asthma.	Pneum. 7 y. ago.	Chills, aching limbs, thirst.	r.	L. base.	Mod. 8 d. Grad.	14 d.	R.	—	
685 Geo. Wilks, M.B.	F.	68	Temp. Suff.	Pneum. Scarlat.	1	1 3	Good. Ind.	?	Delicate, glaucoma 8 y. ago.	Weakness, pain in side.	r.	R. base.	Mild. 5 d. Sud.	10 d.	R.	Severe pain in the muscles of the right iliac region, which lasted about 4	

	F	IS	Temp. Suff.	Tonsill. Bron. Cat.	1	Ind. Ind.	F. laryngo- bron. cat. S. bron. cat.	0	Cough some- times, phth. R. apex.	r.	Both bases.	Sev. till D.	till D.	D. 13thd.
687 Walter Bernard, F.K.Q.C.P.					1	1		0						
688 "	M.	34	Tot. abs. Suff.	Pneum. Catarrh Tonsill. Bron. Cat. Erysip.	2 2 1 2 1	Good. Ind.	F. d. chr. bronch. M. chr. bronch.	Pleurisy.	0	r.	L. base.	Sev. 7 d. Sud.	12 d.	R.
689 G. P. Hadley, M.D.	M.	40	Temp. Suff.	0		Good. Good.	0	Periostit. of os calcis some y ago. ? gout	Pain in side.	r.	R. base.	Mod. 25 d. Grad.	over 25 d.	R.
690 Alfred Macpherson, M.B.	M.	24	Temp. Suff.	Tonsill.	1	Good. Good.	F. chr. bronch.	Tonsill twice yearly, 15-19.	Malaise.	r.	R. base.	Mod. 7 d. Sud.	14 d.	R.
691 "	M.	39	Temp. Suff.	Bron. Cat.	+	Good. Good.	0	0	Weakness.	r.	L. base.	Sev. 8 d. Grad.	21 d.	R.
692 "	F.	17	Temp. Suff.	—		Good. Good.	0	Catarrh, scarlat. last y.	0	r.	Both bases.	Mod. 26 d. Grad.		R.
693 A. MacLean, L.R.C.S.	M.	18	Temp. Suff.	Pneum. Scarlat.	3 +	Good. Good.	M. d. phth. ?	0	Sore throat, catarrh.	r.	Both bases.	Mod. 5 d. Grad.	12 d.	R.
694 F. Walker, M.R.C.S.	M.	21	Temp. Suff.	0	0	Good. Good.	—	0	Pain in side.	r.	R. base. L. whole	Mod. 7 d. Sud.	14 d.	R.
695 "	M.	63	Intemp. Suff.	Pneum.	1	Bad. Ind.	Son pneum.	0	Chills and cough.	r.	R. whole	Sev. 28 d. Grad.	6 m.	R.

Chr. pneum. ending 6 m. later in recover., of face 28th d., abscess over R. nipple still discharging.

1. Sudden onset after fading of measles rash. 2. D. by heart failure. 3. Rapid onset after mental distress. 4. Prolonged mental depression; exposure. 5. Albuminur for 3 d. 6. Prolonged mental strain.



Observer's Name.	Sex.	Age.	Temperate or otherwise. Food.	Concurrent cases of illness in	Same house.	Same district.	Sanitary state of house " " of district	Family history of lung disease.	Previous illnesses of patient.	Premontory symptoms.	Mode of onset.	Part of lungs affected.	Character of fever. Duration of termination	Duration of physical signs.	Result.	Sequelæ.	Note.
696 F. Walker, M.R.C.S.	M.	44	Intemp. Suff.	Pneum.	1		Good. Good.	0	Dysentery, hæmorrhoids.	Faintness, chills, vomiting, pain in side, diarrhoea.	—	R. whole	Mod. 10 d. Grad.	16 d.	R.	0	
697 Thomas Eastes, M.D.	F.	46	Tot. abs. Suff.	Pneum. Scarlat. Bron. Cat. Erysip.	1 1 +	1	Good. Good.	0	Double pneum. rheumatic attacks.	Pain in side.	r.	Both bases.	Mod. 8 d. Sud.	21 d.	R.	R base was inflamed and fol- lowed by pleurisy.	1
698 "	F.	4	Tot. abs. Suff.	Tonsill. Bron. Cat.	2 +	2	Good. Good.	0	0	Malaise.	0	L. apex.	Mod. 4 d. Sud.	2 d.	R.	—	
699 "	M.	21	Intemp. Suff.	Pneum. Scarlat. Bron. Cat. Erysip.	1 1 +	1	Good. Good.	0	0	Aching limbs	r.	L. base.	Mod. 4 d. Grad.	5 d.	R.	—	
700 W. M. Harmer, L.R.C.P.	M.	52	Temp. Suff.	Pneum. Bron. Cat.	1		Good. Good.	Several d. phth.	Bronch. oecas.	—	r.	R. apex.	— Grad.	—	R.	Abscess in R. axilla.	2
701 C. G. M. Lewis, M.R.C.S.	F.	70	Temp. Suff.	Pneum.	2	+	Good. Good.	0	Chr. emphy- sen., gouty joints.	—	—	Both bases.	Mod. 10 d. Grad.	many w.	R.	0	
702 Hugh R. Ker, F.R.C.S.	M.	18	Temp. Suff.	0			Ind. Bad.	B. pleurisy.	Tonsill. twice last y.	0	r.	R. base.	Mod. 5 d. Grad.	15 d.	R.	—	
703 W. J. Mullally, M.D.	M.	36	Temp. Suff.	Pneum. Tonsill. Bron. Cat.	10 few	10 few.	Good. Good.	0	0	Malaise 3 d.	r.	R. base.	Mod. 10 d. Sud.	23 d.	R.	0	
704 Edward Skinner, L.R.C.P.	F.	13	Tot. abs. Suff.	—	1		Good. Good.	?	Mitral dis., cough.	Dyspnoea.	r.	Both bases.	Mild. till D.	till D. 4th d.	D.		

707	"	M.	18	Intemp. Suff.	Tonsill.	+	Bad. Bad.	abs.	—	Pain in side, dyspnea.	—	Both bases.	Mod. 7 d. Grad.	12 d.	R.	—
708	E. J. Wood, M.B.	F.	50	Temp. Suff.	—		Good. Good.	M. } d. phth. 1 S. } 1 B. }	Bronch., neuralgia.	0	r.	R. base.	Mod. 13 d. Grad.	16 d.	R.	0
709	M. G. Biggs, M.R.C.S.	M.	5	Tot. abs. Suff.	Scarlat. Tonsill. Bron. Cat.	4 + +	Ind. Ind.	—	Scarlat. 3 y. ago, <i>Pneum.</i> 2 m. ago.	Malaise.	0	L. base.	Mod. 4 d. Sud.	21 d.	R.	Bronch.
710	Henry N. Holberton, L.R.C.P.	M.	22	Temp. Suff.	0		Ind. Ind.	F. d. phth.	0	Vertigo, headache, sickness.	0	Both bases.	Mild 9 d. Grad.	—	R.	0
711	James Allan, M.D.	M.	43	Temp. Suff.	—		Ind. Ind.	0	Rheum. at 23.	Pain in head, neck, back, swelling R. wrist, dyspnea.	0	Both bases.	Mod. 7 d. Sud.	13 d.	R.	0
712	C. H. Robinson, M.K.Q.C.P.	M.	47	Temp. Suff.	Pneum.	1	Good. Ind.	—	0	—	r.	L. whole	Mod. 11 d. Grad.	16 d.	R.	—
713	F. W. Hum- phreys, L.R.C.P.	M.	20	Temp. Suff.	0		Good. Good.	Phth. in fam.	0	0	—	R. base. L. apex.	Sev. 42 d. Grad.	60 d.	R. part.	Phthisis L. apex.
714	"	M.	50	Temp. Suff.	Bron. Cat.	1	Good. Good.	0	?	Bron. Cat.	0	R. mid.	Mod. 17 d. Grad.	30 d.	R.	0
715	W. Shaw, M.D.	M.	32	Temp. Suff.	Bron. Cat.	2	Ind. Ind.	?	<i>Pneum.</i> last y.	Malaise, anorexia.	0	L. base.	Sev. till D.	till D.	D.	5
716	Chas. Boyce, M.B.	M.	40	Tot. abs. Suff.	Diphth.	3	Ind. Ind.	1 S. d. phth. 23.	0	0	r.	R. apex.	Sev. 8 d. Sud.	14 d.	R.	0
718	"	F.	30	Temp. Suff.	Tonsill. Bron. Cat.	1 1	Ind. Good.	0	0	Shivering, faintness, cough.	r.	R. base. L. whole	Mod. 4 d. Sud.	22 d.	R.	Adhesions at both bases.

1. Syst. apex mur. on 1st d. gone on 3rd d. 2. Inanitic; phys. exam. imposs. 3. T. norm. on 15th d.; got up without permission; relapse on 17th d. lasting 4 d.  
4. Exp. to very hot sun with bare head. 5. D. with failure of heart; rapid edema of R. lung on 8th d. 6. At work in a cesspool at onset.

Observer's Name.	Sex.	Age.	Temperato or otherwise. Food.	Concurrent cases of illness in	Same house.	Same district.	Sanitary state of house " " of district	Family history of lung disease.	Previous illnesses of patient.	Premontory symptoms.	Mode of onset.	Part of lungs affected.	Character of fever. { Duration Termination	Duration of physical signs.	Result.	Sequelae.	Note.
719 J. Jenkin Lloyd, L.R.C.P.	M.	9	Tot. abs. Suff.	Pneum. Tonsill.	1	+	Good. Good.	—	Measles 4 y. ago, <i>pneum.</i> at 1½ y.	Vomiting.	0	R. base.	Sev. 6 d. Sud.	7 d.	R.	—	1
720 W. J. Mullally, M.D.	M.	66	Temp. Suff.	Pneum. Bron. Cat.	1	8 3	Bad. Ind.	0	0	Shivering, pain in side.	r.	R. base.	Sev. till D.	till D.	D.	0	
721 D. G. Crawford, M.B., Surg. A.M.D.	M.	30	Temp. Suff.	Intermit. Remit. in regiment	102	12	Good. Good.	?	Bronch., ague often.	Intermit. fever 4 d.	0	R. whole	Mod. 4 d. Sud.	16 d.	R.	0	
722 "	M.	24	Temp. Suff.	Intermit. Remit. Varicella.	16 2 1	Good. Good.	Good. Good.	?	Ague twice, boil last y.	Cough, pain in side.	0	Both bases.	Mod. 4 d. Sud.	15 d.	R.	0	
723 G. Cecil Dickson, M.B.	M.	36	Intemp. Suff.	Herpes.	1	1	Ind. Bad.	0	<i>Pneum</i> twice, last 3 y. ago.	Pain in side, cough, bilious vomiting.	r.	Both bases.	Mod. till D.	till D.	D.		2
724 R. Ross, L.R.C.P.	M.	3	Temp. Suff.	Pneum. Scarlat. Catarrh. Tonsill. Herpes. Bron. Cat. Erysip.	few. + few. + few. 4	Good. Good.	Good. Good.	0	Bron. sev. oft.; synovitis L. knee; scarlat. with suppurating tonsill. 5 w. ago.	Restlessness, fever, vomiting.	?	L. base.	Sev. 14 d. Grad.	16 d.	R.	0	
725 "	F.	37	Tot. abs. Insuff.	Pneum. Catarrh. Tonsill. Bron. Cat. Erysip.	7	++ + + few.	Ind. Ind.	1 S. bron.	0	Headache, nausea, vomiting.	r.	R. base.	Sev. 12 d. Grad.	Sev. mos.	R.	Acute arthritis R. hip on 9th d., lasting 5 w., abscess burst	3





Observer's Name.	Sex.	Age.	Temperate or otherwise. Food.	Concurrent cases of illness in	Same house.	Same district.	Sanitary state of house " " of district	Family history of lung disease.	Previous illnesses of patient.	Prenatal symptoms.	Mode of onset.	Part of lungs affected.	Character of fever. { Duration Termination } of fever.	Duration of physical signs.	Result.	Sequelae.	Note.
736 J. A. Mackenzie, M.B.	M.	29	Intemp. Suff.	Pneum. Enteric. Scarlat. Catarrh. Tonsill. Bron. Cat. Erysip.	1 1 3 1 2 1	3 few. 1 3 1 2 1	Good. Ind.	0	Otorrhoea 1 m. ago.	Pains, shivering, sickness.	r.	Both bases.	Sev. till D.	till D.	D. 11th d		
737 "	M.	19	Temp. Suff.	Fever. Tonsill. Bron. Cat. Erysip.	1 1 1	1 1 1	Ind. Ind.	B. d. "consumption" at 12.	None for 9 y.	Sore throat, chilliness.	0	L. base.	Mod. 3 d. Sud.	6 d.	R.	0	1
738 W. M. Harman, M.B., Surg. Maj. A.M.D.	M.	38	Intemp. Suff.	Enteric. Folie. Tonsill. Bron. Cat. Ac. bron.	1 1 1 3 5 1	1 1 1 3 5 1	Ind. Ind.	F. d. bron. B. bron.	Pneum. R. 4 y. ago; intermittent.	Feverishness.	r.	R. whole	Sev. 24 d. Grad.	27 d.	R.	—	2
739 J. A. Mackenzie, M.B.	F.	38	Temp. Suff.	Pneum. Catarrh. Tonsill. Bron. Cat. Erysip.	3 3 2 1	3 3 2 1	Good. Ind.	B. bron.	Ac. rheum., bron. yearly. Pneum. L. 5 y. ago.	Shivering, cough, stitch.	r.	R. base.	Mod. 4 d. Sud.	8 d.	R.	0	3
740 "	M.	26	Temp. Insuff.	Pneum. Scarlat. Catarrh. Tonsill. Bron. Cat. Erysip.	3 1 3 2 1	3 1 3 2 1	Bad. Ind.	0	0	Sore throat, weak and ill 3 w.	0	R. base.	Sev. 6 d. Grad.	10 d.	R.	0	
741 John Blair, M.D.	M.	52	Intemp. (very) Suff.	Pneum. Measles. Tonsill.	2 2 2 1	2 2 2 1	Ind. Ind.	M. d. pneum.	0	Out of sorts.	r.	L. base.	Mod. till D.	till D.	D. 3rd d.		
742 J. Mackenzie	M.	22	Temp.	Catarrh.	1	1	Ind.										

744 W. B. Mackay, L.K.Q.C.P.	M.	20	Temp. Suff.	Pneum.	4	Good. Ind.	0	0	Feverish- ness.	0	L. base.	Sev. 42 d. Grad.	28 d.	R.	0	5
745 E. H. Howlett, F.R.C.S.	F.	42	— Suff.	0		Ind. Ind.	0	0	—	r.	R. base.	Mild. Sud.	15 d.	R.	0	
746 E. Maedowel Cosgrave, M.D.	M.	16	Temp. Suff.	—		Ind. Ind.	1 S. d. bron.	0	Cough 3 w.	0	L. base.	Sev. 8 d. Grad.	14 d.	R.	0	
747 James T. George, M.R.C.S.	M	50	Temp. Suff.	Pneum. Enteric. Tonsill. Erysip.	4 + 2 1	Good. Good.	0	Dyspepsia.	0	r.	R. apex.	Sev. 11 d. Grad.	16 d.	R.	0	
748 Henry G. Terry, M.R.C.S.	F.	22	Tot.abs. Suff.	Pneum. Enteric. Tonsill. Erysip.	2 2 1 1	Ind. Ind.	0	Anœmia.	Cough, malaise.	r.	R. base.	Mod. 10 d. Grad.	30 d.	R.	—	
749 C. R. Illing- worth, M.D.	F.	54	Temp. Suff.	0		Good. Good.	0	0	Pain, dyspnoea, cough.	r.	Both bases.	Sev. 30 d. Grad.	60 d.	R.	Bronch. sev.	
750 E. P. Harley, L.R.C.S.	M.	18	Tot.abs. Suff.	0		Good. ?		Scarlat. and rheum. 2 y. ago, <i>Pneum.</i> last y.	Cough, and sore throat 2 w.	r.	L. base.	Mod. 11 d. Grad.	12 d.	R.	—	
751 W. E. Bradley, M.B.	M.	33	Temp. Suff.	Pneum. Scarlat. Tonsill. Herpes. Bron. Cat.	3 5 2 1 +	Good. Good.	0	0	Pain in head and limbs, vomiting.	r.	Both bases.	Sev. 12 d. Grad.	—	R.	0	
752 M. W. Williams, L.R.C.P. (Wheulton).	M.	7	Tot.abs. Suff.	0		Bad. Ind.	0	0	Restlessness, dyspnoea, sore throat.	r.	L. base.	Mod. 11 d. Grad.	8 d.	R.	—	
753 De Vere Hunt, L.R.C.P.	M.	21	Temp. Suff.	0		Good. Good.	M. d. phth.	0	0	r.	L. whole	Sev. 6 d. Sud.	15 d.	R.	0	

1. Inflamed fauces with sloughy suspicious spots. 2. Slight albuminur. in first w.; ague during attack. 3. Slight albuminuria; pregnant 8 m. 4. Unilateral sweating on affected side. 5. Ulceration of soft palate and tonsil.

Observer's Name.	Sex.	Age.	Temperate or other. viso. Food.	Concurrent cases of illness in	Same house.	Same district.	Sanitary state of house " " of district	Family history of lung disease.	Previous illnesses of patient	Pneumatory symptoms.	Mode of onset.	Part of lungs affected.	Character Duration of fever.	Duration of physical signs.	Result.	Sequelae.	Note.
754 R. J. Martin, F.R.C.S.	M.	31	Temp. Suff.	0			Good. Good.	0	Bronch.	0	r.	R. base.	Sev. 3 d. Sud.	10 d.	R.	0	
755 De Vere Hunt, L.R.C.P.	M.	35	Temp. (now). Suff.	0			Ind. Good.	?	Catarhs; con- valesc. from ac. rheum.	0	r.	L. base.	Sev. 5 d. Grad.	11 d.	R.	0	
756 C. M. Jessop, M.R.C.P.E. Brig. Surg. A.M.D.	M.	29	Intemp. Suff.	Pneum. Ague. Tonsill. Bron. Cat.		1 1 2 2	Good. Good.	?	Syph. (prim.) 8 y. ago, debility.	0	r.	L. base.	Sev. 12 d. Grad.	23 d.	R.	0	1
757 G. Deane Bourke, L.K.Q.C.P. Surg. A.M.D.	M.	20	Tot. abs. Suff.	Pneum. Ague. Tonsill. Herpes. Bron. Cat. Erysip.		2 2 3 2 13 1	Good. Good.	0	Chr. rheum.	Headache.	r.	Both bases.	Sev. 9 d. Sud.	16 d.	R.	0	
758 "	M.	20	Temp. Suff.	"			Good. Good.	?	0	Out of sorts 1 w.	r.	R. base.	Sev. 7 d. Sud.	12 d.	R.	0	
759 "	M.	64	Tot. abs. Suff.	Pneum. Ague. Tonsill. Bron. Cat.		1 1 5 7	Good. Good.	0	0	Unwell 1 w. vomiting 1 d.	r.	R. base.	Sev. 8 d. Sud.	—	R.	0	
760 O. Bowen, M.R.C.S.	M.	3	Tot. abs. Suff.	Bron. Cat.	1	?	Good. Good.	M's. M. d. phth., F's. F. d. phth.	Varicella 3 m. ago.	Chilliness, catarrh.	0	L. base.	Sev. 13 d. Grad.	12 d.	R.	0	
761 Duncan J. Mac- kenzie, M.D.	M.	2	Tot. abs. Suff.	Enteric. Tonsill. Bron. Cat.	3	1 1 +	Good. Good.	M's. B. d. phth.	Pertussis } 6 Bronch. } m. ago	Increase of cough.	0	L. base.	Sev. 13 d. Grad.	10 d.	R.	Bronchitis.	
762 C. M. Jessop, M.R.C.P.E.	M.	22	Intemp. Suff.	Pneum. Tonsill.		3 2	Good. Good.	0	?	Vomiting 2d.	r.	R. base, L. whole	Sev. till D.	till D.	D.		2

764 F. J. Allan, M.D.	M.	21	Intemp. Suff.	Catarrh. Bron. Cat.	1	+	+	Ind. Good.	0	0	r.	R. base.	Mod. 8 d. Grad.	18 d.	R.	0	3
765 "	M.	26	Temp. Suff.	Pneum. Enteric. Scarlat. Measles. Catarrh. Tonsill. Bron. Cat. Gland abscess.	1	1	1	Good. Good.	0	0	0	Both bases.	Sev. 36 d. Grad.	—	R. part.	Catarrhal pneum.	
766 James T. W. Baird, M.D.	M.	42	Temp. Insuff.	Pneum. Bron. Cat.	1	+	?	?	0	Ac. rheum.	0	r.	R. base.	Sev. 8 d. Grad.	13 d.	R.	0
767 G. H. Barfoot, M.D.	M.	11	Tot.abs. Suff.	Enteric. Tonsill. Bron. Cat.		1	3	Good. Ind.	F. pneum. twice in last 4 y.	0	0	0	R. base.	Mod. 6 d. Sud.	10 d.	R.	0
768 R. L. Batter- bury, M.D.	F.	15	Temp. Suff.	Pneum. Scarlat.		2	+	Good. Good.	M. chr. bron.	Pertuss. 2 y. ago.	0	r.	Both bases.	Mod. 10 d. Grad.	till D. 32nd d.	D.	
769 "	M.	12	Temp. Suff.	Pneum. Enteric.		2	+	Good. Bad.	M. pleur.	0	0	0	L. base.	Mod. 8 d. Sud.	12 d.	R.	0
770 C. Biddle, L.R.C.P.	F.	27	Temp. Suff.	0				Ind. Good.	0	Epilepsy.	Epilept. fit.	r.	R. base.	Mod. 5 d. Sud.	16 d.	R.	0
771 W. J. Black, L.S.A.	—	7	Tot.abs. Suff.	Pneum. Enteric. Measles. Catarrh. Herpes. Bron. Cat.		2	2	Good. Good.	0	0	0	0	L. base.	Sev. 9 d. Sud.	10 d.	R.	0

1. Lay in snow while drunk; albuminuria first 2 d. 2. Been drinking hard; constant vomiting 5 d. P. M. 3. Albuminuria 1-10th several d.



Observer's Name.	Sex.	Age.	Temperate or otherwise. Food.	Concurrent cases of illness in	Same house.	Same district.	Sanitary state of house " " of district	Family history of lung disease.	Previous illnesses of patient.	Promontory symptoms.	Mode of onset.	Part of lungs affected.	Character of termination } off over.	Duration of physical signs.	Result.	Sequelae.	Note.
772 W. J. Black, L.S.A.	F.	20	Temp. Suff.	Pneum. Enteric. Scarlat. Varicella. Tonsill. Herpes. Erysip.		3 2 2 1 1 1 1	Good. Good.	0	0	0	r.	L. base.	Mod. 9 d. Sud.	21 d.	R.	0	
773 G. H. Blackmore, L.R.C.P.	M.	33	Totabs. Suff.	0			Good. Ind.	0	Rheum.	0	0	L. base.	Sev. 7 d. Sud.	28 d.	R.	0	
774 O. Bowen, M.R.C.S.	M.	6	Totabs. Suff.	Bron. Cat.	1		Good. Good.	M's M. asthma.	Measles } last Pertuss. } y.	Vomiting, purging.	r.	L. base.	Mod. till D. till D.	till D. 12th d	D.	0	1
775 T. J. Burrough, M.D.	M.	26	Temp. Suff.	0			Ind. Good.	0	0	0	r.	R. base.	Sev. 9 d. Grad.	12 d.	R.	0	
776 Arthur Campbell, M.B.	M.	33	Temp. Suff.	0			Ind. Good.	1 S. phth.	Indom. L. lung 14 y. ago.	Vomiting, pain in abd., enough.	r.	Both whole.	Sev. — Grad.	14 d.	R.	0	2
777 J. C. Cartwright, M.R.C.S.	F.	17	Temp. Suff.	Pneum. Bron. Cat.		4 +	Ind. Good.	0	0	Neuralgia, vomiting, abd. pain.	r.	R. base.	Sev. 5 d. Sud.	21 d.	R.	0	
778 J. G. Clendinnen, L.R.C.S.	F.	24	Temp. Suff.	Scarlat. Tonsill.		+	Good. Ind.	0	0	Deafness, headache.	r.	R. base.	Mod. 5 d. Sud.	10 d.	R.	0	3
779 J. T. Collin, M.D.	M.	7	Totabs. Suff.	—			Good. Good.	2 Ch. of same fun. d. bronch.	Measles last y., laryng. strid. in inf.	0	r.	L. base.	Mod. 7 d. Sud.	16 d.	R.	Relapse 2d. aft. crisis lasting 2 d.	
	F.	9	Totabs. Suff.	Scarlat.		4	Good.	0	Scarlat 7 w.		r.	Both	Mod.	18 d.	R.	—	4

M.R.C.S.	Temp. Suff.	Tonsill. Bron. Cat.	1	+	Ind. Ind.	F. asthma. congest. 20.	Pneum. 2 y. ago, thecal abscess 2 w. ago.	0	r.	R. base. L. whole	9 d. Grad.	0
782 W. R. Dambrell Davies, M.R.C.S.	21	Tonsill. Bron. Cat.	1	13 4	Ind. Ind.	F. asthma. congest. 20.	Pneum. 2 y. ago, thecal abscess 2 w. ago.	0	r.	R. base. L. whole	9 d. Grad.	0
783 T. C. Denby, M.R.C.S.	21	Bron. Cat.	1		Ind. Good.	1 S. d. pulm. congest. 20.	Enteric last y.	Malaise.	r.	L. base.	Sev. 7 d. Sud.	0
784 Edwin Fenn, M.R.C.S.	43	0			Good. Good.	0	Bron. Cat.	0	r.	R. base. L. whole	Mod. 6 d. Sud.	R.
785 T. Lambert Hall, M.R.C.S.	16	Diphth. Tonsill. Hortep. Bron. Cat.	+	1 2	Ind. Ind.	0	Syph. 3 m. ago.	0	r.	R. base.	Mod. 5 d. Sud.	0
786 H. Hamilton, M.R.C.S.	23	Pneum. Enteric. Catarrh. Tonsill. Erysip.		3 5 + 1	Ind. Good.	1 S. bron.	0	Headache, languor.	r.	R. base. L. whole	Mod. 9 d. Grad.	R.
787 H. Handford, M.D.	42	?			? Good.	2 S. d. phth.	Enteric 7 y. ago, congest. of lungs 6 m. ago.	0	r.	L. base.	Sev. 11 d. Sud.	R.
788 J. H. Irvin, L.K.Q.C.P.	40	Pneum.		8	Good. Good.	0	Bronch. last y.	0	r.	L. base.	Sev. 9 d. Sud.	0
789 Edwin Jackson, M.R.C.S.	45	Measles. Tonsill. Bron. Cat.		7 1 8	Good. Good.	F. d. phth. 43. M. d. phth. 43. 1 B. phth.	Coughs and colds.	Languor.	r.	R. whole	Mod. till D.	D. 7th d.
790 W. D. Jones, L.R.C.P.	35	Pneum.	2		Ind. Good.	1 B. pleur.	Congest. L. lung twice.	Cough.	r.	R. base.	Sev. — Grad.	R. slow.
791 A. Ransome, M.D.	28	—			Ind. Good.	1 A. d. phth.	Enteric.	—	r.	Both bases.	Sev. 5 d. Grad.	0

1. D. by exhaustion. 2. Exp. to storm; L. lung beginning to resolve when R. lung attacked. 3. Hearing returned sud. with crisis. 4. This was like two attacks, one for each lung, each lasting 5 d., with a crisis between; second attack, for R. lung, very sev. 5. In and out of a boathouse in a cold wind. 6. Case 791 went away leaving 790 at home; 791 was taken ill 6 d. after leaving home, while away; 790 4 d. after her, at home.

Observer's Name.	Sex.	Age.	Temperate or other- wise. Food.	Concurrent cases of illness in	Same house.	Same district.	Sanitary state of house " " of district	Family history of lung disease.	Previous illnesses of patient.	Prenatal symptoms.	Mode of onset.	Part of lungs affected.	Character of fever.	Duration of physical signs.	Result.	Sequelæ.	Note.
792 Charles Love- grove, M.D.	M.	44	Temp. Suff.	Bron. Cat. Erysip.		+ 1	Ind. Good.	0	Ague often.	Chilliness 2 d.	r.	Both bases.	Sev. 14 d. Grad.	12 d.	R.	Phlegmesia, dolens L. leg.	
793 W. E. Luscombe, L.R.C.P.	M.	21	Temp. Suff.	Pneum. Catarrh. Tonsill. Bron. Cat. Erysip.		1 5 2 2 1 1	Ind. Good.	0	0	Catarrh.	r.	R. apex.	Mod. 10 d. Sud.	15 d.	R.	0	
794 M. Mackintosh, M.B.	M.	27	Intemp. Suff.	Scarlat. Catarrh. Tonsill. Herpes. Bron. Cat. Erysip.		8 + 8 + 14 2 2	Ind. Ind.	0	Chr. bron.	Lassitude, anorexia 1 w.	r.	L. base.	Mod. 6 d. Grad.	14 d.	R.	0	
795 Thos. M. Martin, M.D.	M.	28	Temp. Suff.	Pneum.		2	Ind. Good.	0	0	0	r.	L. base.	Mod. 14 d. Grad.	42 d.	R.	—	
796 "	M.	28	Temp. Suff.	Pneum.		2	Good. Good.	0	Tonsill last y.	0	r.	R. base.	Sev. 10 d. Grad.	till D. 70th d	D.	0	1
797 Robert Mears, F.R.C.P.E.	M.	14	Temp. Suff.	Scarlat.		40	Good. Good.	?	Asthma 5 y., congest. R. lung last y.	0	r.	R. whole	Sev. 10 d. Grad.	14 d.	R.	0	
798 John Meredith, M.D.	F.	25	Temp. Suff.	Pneum. Catarrh. Tonsill. Bron. Cat.	1	few. + +	Good. Good.	F. and F.'s 4 S. d. bron.	0	Aching limbs, fatigue, shivering.	r.	L. base.	Mod. 4 d. Grad.	Under 35 d.	R.	—	2
799 T. C. Murphy, L.R.C.P.	M.	72	Temp. —	Bron. Cat.	1		Bad. Good.	0	Bron. often.	Fever, pain in side.	r.	L. base.	Sev. 5 d. Grad.	8 d.	R.	0	
800 A. D. Leith Napier, M.D.	F.	40	Temp. Suff.	Pneum. Catarrh.		3 +	? Good.	0	Asthma many y.	Catarrh.	0	R. base.	Sev. till D.	till D. 5th d.	D.	0	3





Observer's Name.	Sex.	Ago.	Temperate or other-wise. Food.	Concurrent cases of illness in	Same house.	Same district.	Sanitary state of house " " of district	Family history of lung disease.	Previous illnesses of patient.	Premunitory symptoms.	Mode of onsch.	Part of lungs affected.	Character of fever. { Duration Termination	Duration of physical signs.	Result.	Sequelae.	Note.
809 John Reid, M.B.	M.	49	Temp. Suff.	Pneum. Retheln. Measles Catarrh. Tonsill. Herpes. Bron. Cat. Erysip.	3 2 7 4 5 1 12 2	3 2 2 1 5	Good. Good.	—	—	Pain in side, constipation.	r.	R. base.	Mild — Grad.	32 d.	R.	0	
810 W. R. Sergeant, L.R.C.P.	M.	18	Temp. Suff.	Pneum. Catarrh. Tonsill. Bron. Cat.	2 2 1 5	2 2 1 5	Ind. Good.	—	Measles 2 y. ago.	Headache, musc. pain, catarrh.	r.	L. whole	Sev. 6 d. Sud.	14 d.	R.	0	
811 "	F.	30	Temp. Suff.	Pneum. Enteric. Catarrh. Bron. Cat.	1 1 1 3	6 1 1 3	Ind. Good.	0	0	0	r.	Both bases.	Mod. 13 d. Grad.	21 d.	R.	0	1
812 "	M.	30	Temp. Suff.	Pneum. Enteric. Bron. Cat.	3 1 2	3 1 2	? Good.	0	Quinsy twice.	0	r.	Both bases.	Sev. till D.	till D. 10th d.	D.	—	2
813 "	F.	38	Temp. Suff.	Pneum. Catarrh. Bron. Cat.	1 1 1	1 1 1	Good. Good.	M's. S. pneum.	0	0	r.	R. base.	Mod. 9 d. Grad.	16 d.	R.	—	
814 G. A. Sheppard, M.R.C.S. Tom Bates, M.R.C.S.	M.	41	Temp. Suff.	Tonsill. Bron. Cat.	3 2	3 2	Good. Ind.	M. } d. phth. B. } S.	Cough, ex-pectoration and fever, threatening phth. for 6 m. this y.	Siek head-ache, lassitude, chills.	r.	L. whole	Mod. 9 d. Grad.	16 d.	R.	0	
815 T. S. Smith, M.B.	F.	24	Temp. Suff.	--			Ind. Good.	0	0	Headache, pain in side.	—	Both bases.	Sev. 10 d. Sud.	12 d.	R.	0	
816 "	M.	55	Interp. Suff.	Bron. Cat.	3	3	Ind. Good.	—	—	0	r.	L. apex.	Mild till D.	till D.	D. sch d.		3



Observer's	Sex.	Age.	Temperate or otherwise. Food.	Concurrent cases of illness in	Same house.	Same district.	Sanitary state of house " " of district	Family history of lung disease.	Previous illnesses of patient.	Premontory symptoms.	Mode of onset.	Part of lungs affected.	Character of fever. } Duration of physical signs.	Result.	Soquelae.	Note.
828 M. R. J. Behrendt, L.R.C.P.	M.	14	—	—	—	—	—	—	—	Headache.	r.	R. base.	Mod. 5 d. Grad.	R.	—	1
829 "	M.	19	—	—	—	—	—	—	—	Headache and car-ache.	—	L. whole	—	R.	—	
830 "	M.	27	Temp. Suff.	—	—	—	—	—	Weekly, 8 m. pneum. ago.	0	r.	R. base.	Sev. 6 d. Sud.	R.	—	
831 "	M.	—	—	—	—	—	—	—	—	0	r.	L. apex.	Sev. 6 d. Sud.	R.	—	
832 "	F.	—	—	—	—	—	—	—	—	—	—	R. base.	Sev. 7 d. Grad.	R.	—	
833 "	M.	—	—	—	—	—	—	—	—	—	r.	R. base.	—	—	—	2
834 "	M.	8	—	—	—	—	—	—	—	Pain in head and ears.	—	L. base.	—	R.	—	
835 "	M.	43	Interp. (very.)	—	—	—	—	—	—	Vomiting 2 d.	—	R. base. L. apex.	Mild.	D. 1st d.	—	3
836 "	M.	50	Temp. Suff.	—	—	—	—	—	"Pneum. once"	chilly 1 d.	r.	R. apex.	—	R.	—	
837 "	M.	55	Interp.	—	—	—	—	—	—	—	—	R. base.	—	D. 8th d.	—	
838 "	M.	19	—	—	—	—	—	—	—	—	—	R. base.	Sev.	R.	—	
839 "	M.	27	—	—	—	—	—	—	—	—	—	R. apex.	Sev.	R.	—	





Observer's Name.	Sex.	Age.	Temperate or otherwise. Food.	Concurrent cases of illness in	Same house.	Same district.	Sanitary state of house " " of district.	Family history of lung disease.	Previous illnesses of patient.	Premontory symptoms.	Mode of onset.	Part of lungs affected.	Character of fever. Duration Termination	Duration of physical signs.	Result.	Sequelæ.	Note.
S49 C. Biddle, L.R.C.P.	M.	39	Temp. Suff.	Bron. Cat.	1		Good. Good.	1 S. d. "con- sumption."	Hepatic con- gestion.	0	r.	R. base.	Sev. 4 d. Sud.	14 d.	R.	—	
S50 C. Y. Biss, M.B.	M.	12	Tot. abs. Suff.	0			Good. Good.	0	Chorea 2 y. ago, measles 3 y. ago.	Vomiting.	—	R. base.	Sev. 7 d. Sud.	30 d.	R.	0	
S51 M. G. R. Biggs, M.R.C.S.	M.	5	Tot. abs. Suff.	Scarlat. Tonsill.	2 2		Ind. Ind.	0	0	Convulsion, vomiting.	0	R. base.	Sev. 12 d. Sud.	35 d.	R.	0	
S52 J. Blair, M.D.	F.	6½	Tot. abs. Suff.	Pneum. Enteric. Tonsill. Bron. Cat. Erysip.	1 1 1 1		Ind. Ind.	F.'s F. d. ac. pneum. M.'s B. ditto.	Scarlat. } 2 y. Measles } ago.	0	r.	L. base.	Sev. 11 d. Grad.	21 d.	R.	Anthrax on L. buttock.	
S53 W. P. Brabazon, M.D.	M.	13	Temp. Suff.	0			Good. Good.	0	Measles last y.	Ecarache, stiff neck, glands tender.	—	R. whole	Sev. 9 d. Sud.	18 d.	R.	—	
S54 "	M.	10	Temp. Suff.	Pneum.	1		" "	0	Measles last y.	Vomiting, diarrhoea, ecarache, tender neck glands.	r.	Patches of both lungs.	Sev. 9 d. —	18 d.	R.	—	
S55 Essex Bowen, M.D.	M.	50	Temp. Suff.	Pneum. Bron. Cat.	1 6		Good. Good.	?	0	Bilious 2 d.	r.	R. whole	Mod. 9 d. Sud.	16 d.	R.	0	
S56 C. Boyce, M.B.	F.	24	Temp. Suff.	—			Good. Good.	F.'s S. d. phth.	Occas. bilious attacks.	Malaise.	r.	L. whole	Sev. 12 d. Grad.	18 d.	R.	—	
S57 H. Langley Browne, L.R.C.P.	M.	36	Intemp. Insuff.	—			Good. Good.	M. d. phth. 2 S. phth.	Dysent. last y., chron. cough.	—	r.	Both bases and	Sev. 2 Grad.	?	R.	—	

859	"	F.	69	Temp. Suff.	Pneum.	1	"	both sides.	Cellulitis of leg 3 y. ago.	Sickness, headache, fever.	r.	Both wholes.	Sev. till D.	till D. 5th d.	3	
860	W. E. S. Burnett, L. R. C. P.	M.	29	Temp. Suff.	Pneum.	3	Good. Good.	0	0	0	r.	L. base.	Sev. 7 d. Sud.	14 d.	R.	0
861	M. Carmichael, M. D.	F.	52	Tot. abs. Suff.	Tonsill. Herpes.	1 1	Good. Good.	?	Pleur. 5 y. ago, bron. cut. every winter since.	0	r.	R. whole nearly.	Mod. till D.	till D. 8th d.	D.	4
862	T. R. H. Chum, M. R. C. S.	F.	25	Temp. Suff.	0		Good. Good.	?	Ac. rheum., delicate.	Cold, anorexia.	r.	L. apex.	Sev. 9 d. Sud.	14 d.	R.	0
863	"	F.	9½	Tot. abs. Suff.	Pneum. Bron. Cat.	5 3	Good. Good.	0	Bron. Cat.	Nausea, vomiting, malaise, fever, cough.	0	R. base.	Sev. 9 d. Sud.	14 d.	R.	0
864	"	M.	90	Temp. Suff.	Pneum. Bron. Cat.	1 3	Good. Good.	0	Slight bronch.	Cough, pain in side.	0	R. base.	Mod. 4 d. Sud.	10 d.	R.	Slight bronch. debility. 0
865	"	F.	46	Temp. Suff.	Pneum. Bron. Cat. Erysip.	4 + 3	Good. Good.	0	0	0	0	R. base.	Sev. 4 d. Sud.	9 d.	R.	0
866	"	M.	30	Intemp. Suff.	Pneum. Bron. Cat.	1 4 3	Ind. Ind.	1 ch. pneum.	0	Catarrh.	—	R. base.	Mod. 5 d. Sud.	17 d.	R.	0
867	G. M. Colman, M. B.	M.	17	Temp. Suff.	Scarlat. Bron. Cat.	4 1	Bad. Bad.	F. chr. bron., 3 B. and S. d. croup and pertuss.	Conval. from scarlat.	Weakness, headache, anorexia, diarrhoea.	r.	R. whole	Mod. 5 d. Grad.	16 d.	R.	0
868	H. Denme, M. D.	M.	11	Tot. abs. Suff.	Measles. Varicella. Tonsill. Bron. Cat.	1 2 1 4 0	Good. Good.	F's. B. d. phth. 1 S. d. cong. of lungs.	0	Headache.	r.	R. base.	Mod. 7 d. Sud.	11 d.	R.	0

1. In same house; 854 taken ill 5 d. after 853; motions offensive in both; to and fro cardiac sound at base on 3rd d. in case of 854. 2. Abortion at 7th m. on 2nd 1.; slight peritonit. on 4th d. 3. Sisters; 859 nursed 858; 2 d. after her D. was taken ill herself; strong and healthy before; no expos. (Some septic cause; well water bad and drainage defective.—H. L. B.) 4. D. by grad. syncope. 5. 863 was moved from home to house of 864 to escape measles, and caught cold; taken ill 2 d. after; 864 taken ill same d.; measles rash appeared on 863 28 d. after removal. 6. His child was attacked with pneum. 2 d. before him.

Observer's Name.	Sex.	Age.	Temperate or other. wisc. Food.	Concurrent cases of illness in	Same house.	Same district.	Sanitary state of house " " of district	Family history of lung disease.	Previous illnesses of patient.	Premunitory symptoms.	Mode of onset.	Part of lungs affected.	Character (Duration of fever.)	Duration of physical signs.	Result.	Sequelæ.	Note.
869 C. E. Douglas, M.D.	M.	7	Temp. Suff.	Measles. Erysip.		2	Ind. Ind.	F. weak chest, S. pneum.	Scarlat 1 y. ago Measles and otorrhoea 4 y. ago.	Headache, delirium, carache.	—	Both bases.	Sev. 8 d. Sud.	3 d.	R.	—	1
870 G. M. Edmond, M.D.	M.	72	Intemp. Suff.	Pneum. Enteric. Scarlat. Catarrh. Tonsill. Herpes. Bron. Cat. Erysip.	4	2 1 1 + + + + 2	Good. Good.	1 son d. phth.	<i>Pneum. L. base</i> 2 y. ago.	0	r.	L. base.	Mod. 7 d. Grad.	till D. 11thd.	D.	—	
871 "	F.	63	Temp. Suff.	Pneum. Scarlat. Catarrh.		1 + +	Good. Good.	0	0	Pains in bones, mal- aise 1 w.	r.	R. base.	Mod. 8 d. Sud.	21 d.	R.	0	
872 "	F.	64	Temp. Suff.	Tonsill. Herpes. Bron. Cat. Erysip.		2 2 2 +	Good. Good.	M. and M's. fam. all d. phth. 3 S. d. phth.	Bronch. attacks, ac. Bronch. 2 y. ago., heart dilat.	0	r.	R. base.	Mild till D.	till D. 7th d.	D.	—	
873 W. A. Finlay, M.D.	M.	13	Tot. abs. Suff.	Measles. Tonsill. Bron. Cat. Erysip.		1 3 3 1	Good. Good.	0	0	Sickness, headache.	0	R. base.	Sev. 9 d. Grad.	14 d.	R.	0	
874 "	M.	28	Temp. Suff.	Pneum. Varicella. Scarlat. Enteric. Bron. Cat.		2 + 2 2 1 2	Ind. Good.	F. d. phth. 59, 2 B's. d phth. 19 and 21.	Ac. rheum. at 12.	Catarrh 1 w.	r.	R. base.	Sev. 8 d. Grad.	?	R.	0	
875 "	M.	12	Tot. abs. Suff.	Pneum. Tonsill. Bron. Cat.	1	2	Good. Good.	M. d. phth. 41.	Measles 2 y. ago.	Sickness, malaise.	r.	L. base.	Sev. 5 d. Sud.	14 d.	R.	0	
876 G. A. Gibson, M.D.	F.	50	Temp. Suff.	0		3	Good. Good.	2 S. d. phth.	0	Chill 3 d. ago, pain in side.	—	R. base.	Sev. 13 d.	till D. 17thd.	D.	—	2

S78 W. Lamb, M.D.	M.	42	Intemp. Suff.	Catarrh. Herpes. Bron. Cat.	1 1 1	Good. Good.	?	well since.	Malaise, vomiting, diarrhoea.	r.	R. whole nearly.	Sud. Mod. till D.	till D. D. 6th d.	3
S79 "	M.	70	Intemp. Suff.	0		Bad. Ind.	1 B. d. bron.	Pleur. } 7 y. Fits. } ago. Erysip. r. leg 2 y. ago.	Malaise.	r.	R. base.	Mod. 14 d. Grad.	R.	0
S80 S. G. Littlejohn, M.B.	F.	14	Tot. abs. Suff.				?	?	Vomiting.	r.	R. apex.	Sev. 9 d. Sud.	R.	0
S81 "	F.	13	Tot. abs. Suff.					?	Sore throat 2 d.	0	R. apex.	Mod. 5 d. Sud.	R.	0
S82 "	M.	12	Tot. abs. Suff.	Pneum. Measles. Scarlat. Tonsill.	In School.	Good. Good.	?	?	Vomiting 1 d.	0	R. apex. L. base.	Sev. 7 d. Grad.	R.	0
S83 "	M.	11	Tot. abs. Suff.	Herpes. within 6 w.			?	?	Vomiting 1 d.	0	R. base. L. apex.	Mild. 3 d. Sud.	R.	0
S84 "	M.	13	Tot. abs. Suff.				?	?	Vomiting, diarrhoea, pain over L. kidney.	r.	L. apex.	Mod. 5 d. Sud.	R.	0
S85 J. Jenkin Lloyd, L.R.C.P.	F.	37	Temp. Suff.	Pneum. Tonsill. Scarlat.	4 + +	Good. Good.	1 daught., 4, has broncho- pneum.	Peritonitis 11 y. ago.	Pain in side.	r.	R. base.	Mod. 10 d. Grad.	R.	—
S86 "	M.	33	Temp. Suff.	Pneum. Tonsill. Bron. Cat. Scarlat.	1 + + +	Good. Good.	1 daught. pneum. now	Variola.	Tonsill.	r.	L. base.	Mod. 14 d. Grad.	R.	—
S87 J. T. McMahon, L.K.Q.C.P.	M.	29	Temp. Suff.	0		Good. Good.	0	0	0	r.	Both bases.	Sev. 10 d. Grad.	R.	—

1. D. by asthenia. 2. 2 d. aft. convalesce. P. and T. rose very rapidly; next d. coma set in, followed by R. hemipleg.; next d. D. in coma. 3. Was on the verge of del. trem., and had been drinking for 1 m.; intemp. 12 y.; albuminuria 1-5; casts hyaline and granular; liver dulness 2½ inches; spleen much enlarged. 4. Preceded by erysip. R. leg. accomp'd. by erysip. L. leg, both supp.; pseudo-crisis 5th d., worse afterwards; albuminur. 1-5; casts hyaline and granular. 5. The 12 cases fell between Mar. 29 and May 9; school full of illness at the time; cases S80, S81, had abdom. tenderness and diarrhoea, but no erupt.; S82 obstinate bilious vomiting.



Observers	Sex.	Age.	Temperature or other- wise. Food.	Concurrent cases of illness in	Same house.	Same district.	Sanitary state of house " " of district	Family history of lung disease.	Previous illnesses of patient.	Prenatal symptoms.	Mode of onset.	Part of lungs affected	Character of fever.	Duration of physical signs.	Result.	Sequelæ.	Note.
888 Hy. Colley March, M.D.	F.	14	Tot. abs. Suff.	Measles. Enteric. Scarlat. Catarrh. Tonsill. Herpes. Bron. Cat. Erysip.	1	3 2 2 3 2 3 2 3	Good. Good.	M's S.d. phth., cousin d. hydro-ceph.	0	Earache, languor and excitement alternately.	r.	L. base.	Sev. 9 d. Sud.	30 d.	R.	0	1
889 "	—	—	—	Pneum. Measles. Scarlat. Catarrh. Tonsill. Herpes. Bron. Cat.	1	3 + + + 2 6 3 3	Good. Good.	1 daugh. bronch. attacks.	Ecz. of hand.	0	r.	R. mid. L. base.	Mod. 8 d. Sud.	12 d.	R.	0	
890 W. W. Millard, M.B.	M.	7½	Tot. abs. Suff.	Scarlat. Bron. Cat. Erysip.		+	Good. Good.	0	Ac. tonsill last y., macules 5 m. ago.	Headache, cough.	r.	R. base.	Sev. 12 d. Grad.	16 d.	R.	Diarrhœa like ty- phoid set in on 27th d.	
891 John W. Moir, M.D.	M.	38	Intemp. Insuff.	Pneum. Tonsill. Bron. Cat. Erysip.		2 6 2 1	Bad. Good.	0	0	0	r.	L. base.	Sev. 8 d. Sud.	Over 13 d.	R.	0	
892 "	M.	44	Intemp. Insuff.	Tonsill. Bron. Cat. Erysip.		1 3 1	Ind. Ind.	F. d. phth. 40.	Relapsing fev. 12 y. ago, chest troubles ever since.	0	r.	L. base.	Mod. 7 d. Grad.	—	R.	Tubercular consolid. mid. of R. lung.	
893 W. S. Paget, M.D.	M.	32	Tomp. Suff.	0			Ind. Good.	0	0	0	r.	R. whole L. base.	Sev. till D. till D.	till D.	D.		2
894 Herbert Parsons, M.R.C.S.	M.	56	Intemp. Suff.	Catarrh.	4	1	Good. Good.	0	Pneum. L. 20 y. ago, superficial abscess in chest walls	Lassitude, anorexia.	r.	L. base.	Sev. till D. till D.	till D.	D. 6th d.		3

893 John Rand, M.B.	M.	12	Temp. Suff.	0	Good. Good.	F. } B. }	Bron. Cat.	Vomiting.	r.	R. base.	Mon. 7 d. Grad.	21 d.	R.	9
896 H. G. Rawdon, M.D.	F.	21	Tot. abs. Suff.	0	Good. Good.	F. } B. }	Delicate 2 y. ago.	—	0	R. base.	Sev. till D.	till D.	D. 5th d.	
897 John Reid, M.B.	M.	33	Temp. Suff.	Pertuss. Rubeola. Tonsill. Herpes. Bron. Cat. Erysip.	3 2 2 2 6 1	Bad. Good.	0	Pleuritic pains, run- bling.	r.	L. base.	Sev. 5 d. Sud.	6 d.	R.	0
898 Duncan J. Reid, M.B.	M.	37	Tot. abs. Suff.	Typhus. Enteric.	+ +	Good. Good.	0	Headache, malaise.	r.	R. base.	Mod. 25 d. Grad.	Cver 27 d.	R. part.	Chron. pneum. of both apices behind.
899 Frank Salter, M.R.C.S.	F.	35	Temp. Insuff.	Scarlat. Erysip.	2 1	Ind. Ind.	0	Cold, mal- aise, dys- pnoea.	r.	R. base.	Mod. 4 d. Grad.	10 d.	R.	
900 W. D. Sheppard, L.R.C.P.	M.	22	Temp. Suff.	—	Ind. Ind.	Ind. Ind.	M. d. phth. F. d. bronch.	0	r.	R. base.	Sev. 8 d. Grad.	14 d.	R.	0
901 "	M.	90	Intemp. Suff.	Erysip.	1	Ind. Ind.	F. d. bronch.	0	r.	L. base.	Mod. 8 d. Sud.	—	R.	
902 J. Taylor, F.R.C.S.	F.	11	Temp. Suff.	Enteric. Tonsill. Bron. Cat.	3 2 +	Good. Good.	0	0	0	L. base.	Mod. 4 d. Sud.	10 d.	R.	0
903 T. Edgar Under- hill, M.B.	F.	40	Temp. Suff.	Pneum. Scarlat.	6 +	Good. Ind.	0	Headache, fever.	r.	R. base.	Mod. 7 d. Sud.	28 d.	R.	0
904 "	M.	63	Temp. Suff.	Pneum. Scarlat. Tonsill. Bron. Cat.	3 2 2 3	Good. Good.	0	Chilliness.	r.	Both bases.	Mod. till D.	till D.	D. 7th d.	4
905 "	M.	32	Intemp. Suff.	Pneum. Scarlat. Tonsill. Bron. Cat.	4 + 1 2	Ind. Ind.	0	Pain in side, dys- pnoea.	r.	Both bases.	Sev. till D.	till D.	D. 6th d.	5

1. Slight rheumat. also. 2. D. by asphyxia. 3. D. by syncope. 4. T. fell on 5th d., seemed convalesce; R. side only affect; that d. wind changed to N.E., L. side attacked, T. rose again; D. by syncope. 5. Drinking heavily for months.

Observer's Name.	Sex.	Age.	Temperate or other. wise. Food.	Concurrent cases of illness in	Same house.	Same district.	Sanitary state of house " " of district	Family history of lung disease.	Previous illnesses of patient.	Prodromatory symptoms.	Mode of onset.	Part of lungs affected.	Character Duration Termination of fever	Duration of physical signs.	Result.	Requiesc.	Note.
906 T. Edgar Underhill, M.B.	F.	20	Tot. abs. Suff.	Pneum. Scarlat. Enteric. Bron. Cat. Erysip.		3 + + + +	Good. Good.	F. d. phth. 40.	0	Fever, thirst, pain in side.	r.	L. base.	Sev. 8 d. Grad.	17 d.	R.	Hæmopt. once since. No signs.	
907 Thos. J. Webster, M.R.C.S.	M.	boy.	— Suff.	Pneum. Catarrh. Bron. Cat.		4 + +	Good. Good.	M's. fam. phth.	Scarlat 2 y. ago	0	r.	L. base.	Sev. 6 d. Sud.	—	R.	—	
908 "	M.	33	— Suff.	Pneum. Catarrh. Bron. Cat.		4 + +	Good. Good.	F. d. hæmopt. 66.	Colic 1 y. ago.	0	r.	L. base.	Sev. 8 d. Grad.	—	R.	—	
909 "	M.	21	Temp. Suff.	Rötheln. Tonsill. Bron. Cat.		5 1 1	Ind. Good.	0	Measles 6 y. ago.	0	r.	R. apex.	Sev. 10 d. Grad.	16 d.	R.	0	
910 D. M. Williams, L.K.Q.C.P. (Liverpool.)	M.	59	Tot. abs. Suff.	—			Good. Good.	0	Pneum. R. 40 y. ago. Pneum. L. 20 y. ago.	0	r.	R. base.	Mod. 6 d. Sud.	18 d	R.	0	
911 O. Williams, L.K.Q.C.P. (Rhosygaer.)	M.	38	Tot. abs. Suff.	Herpes.		1	Ind. Good.	0	0	Headache, lassitude, thirst, sickness.	r.	R. base.	Mod. 9 d. Grad.	22 d.	R.	Pain R. side occas.	
912 T. Wollaston, M.D.	F.	40	Temp. Suff.	Tonsill. Bron. Cat. Erysip.	4 1	2 3 2	Good. Good.	—	Freq. Tonsill. Uterus dis- placed and ulcerated.	Catarrh.	r.	R. base.	Sev. 12 d. Grad.	24 d.	R.	0	
913 F. H. Alderson, M.D.	M.	23	Temp. Suff.	Erysip.		+	Ind. Ind.	F. d. pneu m. 1 S. "con- sumptivo."	0	Chilliness, dyspnea, pain in side.	0	Both bases.	Sev. 7 d. Grad.	10 d.	R.	0	
914 W. F. Brook, M.R.C.S.	M.	46	Temp. Insuff.	0			Good. Good.	0		0	r.	Both bases.	Mod. — Grad.	14 d.	R.	—	





Observer's Name.	Sex.	Age.	Temperate or other- viso. Food.	Concurrent cases of illness in	Same house.	Same district.	Sanitary state of house " " of district	Family history of lung disease.	Previous illnesses of patient.	Prodromitory symptoms.	Mode of onset.	Part of lungs affected.	Character Duration of fever.	Duration of physical signs.	Result.	Sequelæ.	Note.
927 J. Jenkin Lloyd, L.R.C.P.	F.	24	Temp. Suff.	Tonsill.		+	Good. Good.	0	0	Headache.	0	Both bases.	Sev. 29 d. Grad.	Over 44 d.	R. part.	—	
928 "	M.	18	Temp. Suff.	Pneum. Tonsill. Bron. Cat.		4 ++ ++	Good. Good.	0	0	Headache.	r.	Both bases.	Mod. 7 d. Grad.	26 d.	R.	—	
929 "	M.	27	Intemp. Suff.	Tonsill. Bron. Cat.		+	Ind. Bad.	0	<i>Pneum. L.</i> twice. <i>Pneum. R.</i> 3 y. ago. <i>Pneum. R.</i> y. ago.	Pain in side.	r.	L. base.	Mod. 10 d. Grad.	13 d.	R.	—	
930 "	M.	19	Temp. Suff.	Tonsill. Bron. Cat.		+	Good. Good.	M. pleur.	—	Shivering.	r.	Both bases.	Mod. over 17 d.	Over 17 d.	R.	—	
931 "	M.	22	Intemp. Suff.	Pneum. Tonsill. Bron. Cat.		6 ++ ++	Ind. Bad.	B. Pneum.	—	Pain in side.	r.	Both bases.	Mod. 12 d. Grad.	20 d.	R.	—	1
932 W. Odell, F.R.C.S.	M.	9	Temp. Suff.	Pneum. Rötheln.		3 3	Bad. Ind.	0	Measles 3 y. ago, scarlat. last y.	Sickness, headache.	r.	Both bases.	Sev. 3 d. Sud.	6 d.	R.	0	
933 Geo. D. Powell, M.D.	F.	48	Intemp. Suff.	Pneum. Measles.		++	Good. Ind.	0	Del. trem.	—	r.	R. whole	Sev. 21 d. Grad.	63 d.	R.	Debility.	2
934 G. A. Rae, L.R.C.P.	F.	36	Temp. Suff.	Pneum. Measles. Catarrh. Tonsill. Bron. Cat. Erysip.		2 ++ 4 ++ ++ 1	Good. Good.	M. } d. phth. B. } S. }	Bron. Cat.	0	r.	Both bases.	Mod. till D.	till D. 13th d.	D.		3
935 Rich. Rice, M.R.C.S.	F.	15	Temp. Suff.	Scarlat. Horpes.		2 1	Ind. Ind.	0	Scarlat. last y., tonsill off.	Malaise, pain in side.	0	L. whole	Mod. 34 d. Grad.	21 d.	R.	0	
936 Fred. T.	M.	54	Temp.	Pneum		5	Good.	1 B. d. phth.	Tonsill.	0	r.	R. base.	Mod.	21 d.	R.	—	

	Scariat. Tonsill. Bron. Cat.	+ 3 +	Ind. Ind.	F. d. pneum. 29.	2 y. ago and 4 m. ago.	Cold, headache.	r. L. apex.	5 d. Grad.	0
938 J. W. Wolfenden, L.R.C.P.	Temp. Suff.	25	3 2 4	Ind. Ind.	0	Cold, headache.	r. L. apex.	Sev. 7 d. Sud.	10 d. R.
939 "	Temp. Suff.	21	2 10 3	Ind. Ind.	0	Headache, cold.	r. R. apex.	Sev. 5 d. Sud.	0 R.
940 C. H. Watts Parkinson, M.R.C.S.	Temp. Suff.	35	1 1 1	— Ind.	Ac. rheum. 20 y. ago.	—	Both bases.	Mod. 14 d. Grad.	Over 36 d. R. part.
941 Robt. Park, M.D.	Temp. Suff.	34	+	Good. Ind.	Pneum. L. 3 y. ago.	Malaise.	r. L. base.	Mild 14 d. Grad.	Over 24 d. R. part.
942 "	Temp. Suff.	54	+	Good. Good.	Pleur. L. 6 m. ago.	0	0 L. base.	Mild 7 d. ? Grad.	Over 12 d. R. part.
943 T. F. Pearce, M.D.	Tot. abs. Suff.	6	1	Bad. Ind.	0	Sickness, soreness.	r. R. base.	Mod. ? Sud.	4 d. R.
944 "	Tot. abs. Suff.	13	3	Ind. Ind.	Weakly.	Headache, sickness.	0 R. base.	Mod. 7 d. Grad.	9 d. R.
945 G. T. Schofield, L.R.C.P.E.	Temp. Suff.	48	Good. Good.	F. d. chr. bron. S. d. phth. 33.	Enteritis 30 y. ago, dys- pepsia.	Pains R. apex, dyspnoea.	r. Both whole.	Mod. till D.	till D. D. 10th d.
946 J. Ingleby Mac- kenzie, M.B.	Temp. Suff.	3½	Ind. Good.	0	0	—	r. Both bases.	Mod. 8 d. Sud.	9 d. R.
947 J. W. Gooch, M.R.C.S.	Temp. Suff.	64	Good. Good.	0	0	Pain in side, dyspnoea, headache.	r. R. base.	Mod. 5 d. Grad.	14 d. R.

1. 2 sudden deaths from pneum., each on 1st d., close to this house a few d. before onset of 931. 2. During an attack of "black" measles, 3. D. by exhaustion.

Observer's Name.	Sex.	Age.	Temperate or other-wise. Food.	Concurrent cases of illness in same house.	Same district.	Sanitary state of house " " of district	Family history of lung disease.	Previous illnesses of patient.	Premontory symptoms.	Mode of onset.	Part of lungs affected.	Character of fever { Duration Termination	Duration of physical signs.	Result.	Sequelae.	Note.
948 J. Mulvany, M.D.	F.	7	Tot. abs. Suff.	0		Good. Ind.	F. pneum.	0	Sickness, drowsiness.	r.	L. base.	Mild 3 d. Sud.	4 d.	R.	Submax. gland inflam.	
949 Duncan J. Mackenzie, M.D.	M.	8	Tot. abs. Suff.	Bron. Cat. Erysip.	4 1	Good. Ind.	0	Colds.	Chilliness, pallor.	r.	L. base.	Mild 4 d. Sud.	6 d.	R.	0	
950 J. Mackenzie Booth, M.D.	M.	10	Tot. abs. Suff.	Catarrh. Tonsill. Bron. Cat. Enteric.	3 5 1 +	Ind. Bad.	2 A's. } d. 1 U. } phth. M. phth.	0	Cough, sore throat.	r.	L. base.	Mod. 8 d. Grad.	—	R.	—	
951 J. Walters, M.B.	F.	46	Tot. abs. Suff.	Tonsill.	1	Ind. Good.	Child pneum.	Enteric 7 y. ago, <i>Pneum.</i> 4 y. ago, gallstone and peritonitis 2 y. ago	Cold, sickness.	r.	L. base.	Mod. 5 d. Sud.	14 d.	R.	—	
952 E. G. A. Walker, M.R.C.S.	F	7	Tot. abs. Suff.	Tonsill.	1	Good. Good.	M's B. } d. M's S. } phth. F's B. } S. pneum.	Measles 3 y. ago.	Shivering, cold.	—	R. apex. L. base.	Sev. 8 d. Grad.	14 d.	R.	—	
953 T. W. H. Garstang, M.R.C.S.	M.	19	Temp. Suff.	Pneum. Enteric. Tonsill. Bron. Cat.	2 4 1 1	Good. Good.	0	0	Vomiting, malaisc.	r.	R. whole	Sev. 8 d. Grad.	14 d.	R.	0	
954 J. G. Clendinnen, L.R.C.S.	M.	—	Temp. Suff.	Scarlat.	1	Bad. Bad.	B. d. phth	0	Cough.	r	R. whole	Mild till D. till D.	till D. 5th d.	D.		1
955 C. Firth, M.B.	F.	28	Temp. Suff.	0		Ind. Ind.	?	0	Pain.	r.	Both bases.	Mod. till D. till D.	till D. 9th d.	D.		2
956 J. Farrant Fry, L.R.C.P.	M.	8	Tot. abs. Suff.	Pneum. Measles. Catarrh. Tonsill.	2 4 2 1 +	Good. Good.	0	0	Vomiting.		R. whole L. base.	Mod. 28 d. Grad.	over 31 d.	R. part.	R. empyema discharging into bronchus	

L.R.C.P.	M.	Suff.		Good.	M. d. phth. Son ditto.	Chr. rheum.	of throat.	bases.	till D.	8th d.	
958 Thomas Fielding, M.D.	65	Temp. Suff.	—	Good. Good.			Vomiting.	R. whole L. base.	Mod. 12 d Grad.	R.	5
959 F. B. Mallet, M.D.	37	Temp. Suff.	0	Good. Good.	0	0	Shivering, pain in chest.	Both bases.	Sev. 9 d. Grad.	R.	6 Pain in L. side.
960 H. Conpland, Taylor, M.D.	26	Temp. Suff.	Pneum. Bron. Cat.	2 3 Good. Good.	0	0	Bilious, vomiting, pain in side.	R. base.	Sev. till D.	D. 5th d.	7
961 G. H. Whitaker, L.R.C.P.	1}	Tot. abs. Suff.	Pneum. Bron. Cat. Inf. Remit.	+ 2 Good.	?	Pneum. 9 m. ago, diarr. rheo 1 w.	Cerebral dis- turbance, pain in head.	L. base.	Sev. till D.	D. 6th d.	8
962 P. Caldwell Smith, M.B.	38	Temp. Suff.	Pneum. Tonsill. Bron. Cat. Erysip.	2 3 6 1 Ind. Bad.	0	<i>Infl. some lung</i> 24 y. ago.	0	L. base.	Mild 5 d. Grad.	R.	0
963 C. E. Shelly, M.B.	13	Temp. Suff.	Pneum. Catarrh. Tonsill. Bron. Cat. Erysip.	5 5 13 3 11 2 Good. Ind.	0	0	Diarrhoea 3 d.	R. base.	Mod. 5 d. Sud.	R.	0
964 Wm. Sellers, Jun., M.B.	22	Temp. Suff.	0	Good. Good.	0	0	Cough, stitch, fever.	R. base.	Sev. 7 d. Sud.	R.	10 —
965 L. Drnutt, M.D.	24	Temp. Suff.	Pneum. Bron. Cat. Erysip.	3 + 2 Bad. Ind.	0	0	Pains all over, shivering, cough.	Both bases.	Mod. 8 d. Grad.	R.	0
966 E. O. Daly, M.B.	19	Temp. Suff.	0	Ind. Ind.		Syph. 6 m. ago.	Headache, dullness.	R. base.	Mod. 6 d. Sud.	R.	0

1. No hope from onset; extr. prostrat.; D. by failure of heart. 2. Attrib. to sleeping in draught; P. M. exam. 3. L. base was recovering when R. affected. 4. Rigor 17th Jan.; on 18th ac. indam. of throat, not of tonsils (supposed either gouty or erysip.); on 21st lungs affected, R. elbow and arm inflamed, throat relieved; urine highly albumin; due to blood disorder rather than damp or cold. 5. R. lung began to resolve on 6th d.; L. lung attacked on 6th d., began to resolve on 12th d. 6. Due to cold. 7. Miscarriage at 6th m. on 3rd d. 8. Meningitis also; D. by convulsions. 9. Cong. of liver; bile pigments in urine 3 d. 10. Sat in wet clothes; rigor 24 h. after the wetting.



Observer's Name.	Sex.	Age.	Temperate or otherwise. Food.	Concurrent cases of illness in	Same house.	Same district.	Sanitary state of house " " of district	Family history of lung disease.	Previous illnesses of patient.	Premonitory symptoms.	Mode of onset.	Part of lungs affected.	Character of fever. Duration of fever.	Duration of physical signs.	Result.	Sequelae.	Note.
967 H. Denne, M.D.	M.	18	Temp. Suff.	Enteric. Scarlat. Measles. Variola. Catarrh. Tonsill. Bron. Cat.		1 2 3 4 5	Good. Good.	0	0	Vomiting, headache.	r.	L. base.	Mod. 6 d. Grad.	10 d.	R.	0	1
968 James Edwards, L.R.C.P.	F.	18	Tot. abs. Suff.	—			Ind. Good.	3 M's. B's. d. phth., M. chr. bronch.	Bron. cat. each winter.	Cough.	0	R. base.	Mild few d. Grad.	48 d.	R.	0	
969 George Beatson, M.D.	F.	32	Temp. Suff.	0			Good. Good.	0	Dyspepsia, convalesce. fr. influenza.	0	r.	Both bases.	Sev. 7 d. Sud.	28 d.	R.	0	2
970 Essex Bowen, M.D.	M.	26	Temp. ? Suff.	0			Good. Good.	0	0	Pain in side.	0	R. base.	Mod. 10 d. Grad.	17 d.	R.	0	
971 A. de W. Baker, L.R.C.P.	M.	50	Intermp. Suff.	0			Good. Good.	?	Weak heart, muse. tremors.	Pain in side, nervousness, liver derangement.	0	R. whole	Mod. till D. till D.	till D. 3rd d.	D.		3
972 Wm. Berry, L.R.C.P.	F.	19	Temp. Suff.	0			Good. Good.	0	0	Chilliness, malaise } 2d.	r.	R. base.	Mod. 7 d. Grad.	10 d.	R.	0	
973 C. Biddle, L.R.C.P.	F.	44	Temp. Suff.	0			Good. Good.	M. d. phth. 1 Son d. measles and bronch.	0	0	r.	L. whole	Mod. 14 d. Grad.	Over 33 d.	R.	0	4
974 Macfie Campbell, M.D.	M.	24	Tot. abs. Suff.	Pneum. Scarlat. Catarrh. Tonsill. Bron. Cat.		2 1 1 1 3	Good. Good.	M. pleur., 3 B's. bronch. 2 B's. pneum.	—	Shivering, restlessness, cough	r.	R. base.	Sev. 10 d. Grad.	Over 32 d.	R.	—	

	L.R.C.P.	Age	Sex	Temp.	Herpes. Bron. Cat. Erysip.	1 +	Good. Ind.	1 B. } 1 S. }	tropics.	Malaise.	r.	to apex. L. base.	8 d. Grad.	
976	J. G. Clendinnen, L.R.C.S.	M.	28	Temp. Suff.	—	1 +	Good. Ind.	0	Pleur. 5 y. ago, cough 2 y.	Malaise.	r.	R. base.	Mod. till D.	6
977	A. Hamilton, L.R.C.P. (Chester).	M.	25	Intemp. Suff.	Pneum. Enteric. Tonsill. Erysip. Indueuz.	2 7 + 1 +	Ind. Ind.	0	0	Cough 3 w.	r.	R. base.	Sev. 15 d. Grad.	0
978	"	M.	45	Intemp. Suff.	Pneum. Enteric. Tonsill. Erysip. Indueuz.	2 11 + 2 +	Ind. Ind.	0	0	Influenza.	—	R. whole	Sev. 9 d. Sud.	—
979	James Neil, M.D.	F.	75	Temp. Suff.	Catarrh. Tonsill. Bron. Cat.	+	Good. Good.	?	?	Bron. cat.	0	L. whole	Mod. till D.	7
980	Thos. J. Oller- head, L.R.C.P.	F.	72	Temp. Suff.	Catarrh. Tonsill. Bron. Cat.	+	Good. Good.	0	Rheum. gout.	—	?	Both bases.	Mod. till D.	8
981	Herbert Sloman, L.R.C.P.	M.	9	Temp. Suff.	Diphth. Diarrh. Catarrh. Tonsill. Bron. Cat.	6 8 4 21 20	Ind. Ind.	—	—	Catarrh, vomiting 1 d.	r.	Both apices.	Mod. 6 d. Grad.	9
982	"	M.	12	Temp. Insuff.	Pneum. Diphth. Pertuss. Tonsill. Scarlat. Bron. Cat. Erysip.	2 5 60 14 3 36 2	Ind. Ind.	—	—	Weakness, malaise, cold, vomiting.	r.	R. apex.	Mod. 8 d. Grad.	
983	S. D. Clipping- dale, M.D.	F.	70	Intemp. Suff.	—		Good. Good.	—	<i>Infl. R. lung</i> 2 y. ago.	Lassitude, anorexia.	—	R. base.	Mod. 4 d. Grad.	—
984	H. O. Stuart, M.R.C.S., Surgeon A.M.D.	M.	21	Temp. Suff.	Pneum. Enteric. Tonsill. Bron. Cat.	+	Ind. Ind.	—	Alveolar abscess 1 y. ago.	Pain in side, cough.	r.	L. base.	Sev. 4 d. Sud.	

1. Prob. caught on a long journey. 2. Soft mitr. mur. during attack. 3. Lived chiefly upon alcohol; hopeless from first. 4. Due to cold. 5. From mid. to apex, then down; extreme base escaped. 6. Exp. to great heat in furnace yard. 7. Chronic dementia; P.M. examination. 8. D. by failure of heart. 9. Relapse on 12th d., R. apex affected, lasting 3 d.; ditto on 19th d., R. mid. affected, lasting 5 d.

Observer's Name.	Sex.	Age.	Temperate or otherwise. Food.	Concurrent cases of illness in	Same house.	Same district.	Sanitary state of house " " of district	Family history of lung disease.	Previous illnesses of patient.	Premontory symptoms.	Mode of onset.	Part of lungs affected.	Character Duration Termination of fever.	Duration of physical signs.	Result.	Sequelæ.	Note.
985 P. W. MacDonald, M.B.	M.	—	Temp. Suff.	Bron. Cat.	2		Good. Good.	—	Similar attack, syst. aort. mur.	0	0	L. base. R. patches.	Mild. 5 d. Sud.	4 d.	R.	Dyspnoea worse.	1
986 A. O. Grosvenor, M.D.	F.	36	Temp. Suff.	—			Ind. Good.	F. d. phth. S. phth. M. asth.	Ac. rheum. 1 y. ago.	Chills 1 w.	r.	Both bases.	Sev. 5 d. Sud.	10 d.	R.	0	
987 J. Breward Neal, M.R.C.P.E.	F.	58	Temp. Suff.	—			Good. Good.	0	Infl. leg 7 y. ago.	Sudden prostration, epistaxis.	0	R. whole	Sev. ? Grad.	30 d.	R.	—	
988 M. K. Hargreaves, M.B.	F.	29	Intemp. Insuff.	Pneum. Scarlat. Tonsill. Erysip.		4 15 3 1	Ind. Good.	0	Dyspepsia.	Dyspnoea, weakness, fever, anorexia.	r.	R. whole L. base.	Sev. 11 d. Sud.	till D. 16th d	D.		2
989 "	F.	32	Temp. Suff.	Pneum. Scarlat. Bron. Cat.		3 9 12	Good. Good.	M. bronch. Fam. subj. to lung dis.	Cough for 4 winters.	Malaise, sickness, dyspnoea.	r.	R. base. L. whole	Mod. 6 d. Grad.	10 d.	R.	Bronch. as before.	
990 D. O. Fontaine, L.R.C.P.	F.	19	Temp. Suff.	0			Ind. Good.	0	Miscarriage 4 d. before.	Pain in chest, vomiting.	r.	Both bases.	Sev. till D.	till D. 16th d	D.		3
991 James Taylor, L.R.C.P.	M.	16	Temp. Suff.	Enteric. Bron. Cat.		7 +	Good. Good.	0	0	Pain in head and back, chilliness 3 d.	0	R. base.	Mod. 21 d. Grad.	28 d.	R.	0	
992 Arthur Sutherland, M.B.	M.	58	Temp. Insuff.	Pneum. Catarrh. Tonsill. Bron. Cat.	1	4 + + +	Ind. Good.	0	0	Chilliness.	r.	Both bases.	Sev. till D.	till D. 28th d	D.		4
993 A. Cowley-Malley, M.B.	M.	17	Temp. Suff.	Pneum. Ac. Rheum. Tonsill. Herpes.		4 1 2 1	Good. Good.	0	0	—	r.	Both bases.	Sev. 6 d. Sud.	22 d.	R.	0	





Observer's Name.	Sex.	Age.	Temperate or otherwise. Food.	Concurrent cases of illness in	Same house.	Same district.	Sanitary state of house " " of district	Family history of lung disease.	Previous illnesses of patient.	Premontory symptoms.	Mode of onset.	Part of lungs affected.	Character of fever. Duration of fever.	Duration of physical signs.	Result.	Sequelæ.	Note.
1003 A. J. Allott, M.D.	M.	50	Temp. Suff.	—			Good. Good.	0	—	Catarrh.	r.	L. base.	Mod. till D.	till D. 4th d.	D. 4th d.		1
1004 C. Broomhead, M.D.	M.	6	Tot. abs. Suff.	Scarlat. Tonsill. Erysip.		2 2 1	Bad. Ind.	M. } F.'s B. } pneu.	Measles 2 y. ago, bronch. 4 y. ago, convuls. in inf.	0	r.	L. base.	Sev. 6 d. Grad.	14 d.	R.	0	
1005 "	M.	22	Temp. Suff.	Pneum. Scarlat. Enteric. Tonsill. Bron. Cat. Erysip.		1 1 1 1 2 1	Good. Ind.	M. d. bron.	0	0	r.	R. base.	Sev. 8 d. Grad.	15 d.	R.	0	
1006 H. Langley Browne, L.R.C.P.	M	16	Temp. Suff.	—			Good. Good.	U. d. pneum. 46.	0	Vomiting, fever, delirium, pains in back and limbs.	r.	R. base.	Sev. 4 d. Grad.	5 d.	R.	0	
1007 Charles Boyce, M.B.	M.	5	Tot. abs. Suff.	—			Good. Good.	—	Capill. bronch. 3 y. ago.	Stomach de- rangement.	—	Both bases.	Sev. 7 d. Sud.	9 d.	R.	0	
1008 "	M.	51	Temp. Suff.	—			Good. Good.	0	Rheum. gout twice.	Gastric dis- turbance.	r.	L. base.	Sev. 6 d. Sud.	11 d.	R.	0	
1009 "	F.	53	Temp. Suff.	—			Good. Good.	M. d. phth.	Hæmopt. three times.	Catarrh, stomach derange- ment.	r.	L. base.	Mod. 6 d. Sud.	10 d.	R.	Weakness.	
1010 John E. Allen, L.R.C.P.	M.	16	Tot. abs. Suff.	Scarlat. Tonsill. Bron. Cat.		3 1 2	Ind. Ind.	F. d. phth. B. chr. bron. F.'s 2 S. d.	Scarlat. 7 y. ; ago.	0	r.	R. base.	Sev. 8 d. Grad.	12 d.	R.	0	



Observer's Name.	Sex.	Age.	Temperate or other- viso. Food.	Concurrent cases of illness in	Same house.	Same district.	Sanitary state of house " " of district	Family history of lung disease.	Previous illnesses of patient.	Premontory symptoms.	Mode of onset.	Part of lungs affected.	Character Duration Termination of fever	Duration of physical signs.	Result.	Sequelæ.	Note.
1022 A. D. Leith Napier, M.D.	M.	22	Temp. Suff.	Pneum. Scarlat. Catarrh. Tonsill. Bron. Cat. Erysip.	4 4 14 2 2 6	4 4 14 2 2 6	Ind. Good.	0	0	Feverish, catarrh.	0	R. base.	Mod. 7 d. Grad.	10 d.	R.	0	1
1023 "	F.	21	Temp. Suff.	Pneum. Scarlat. Rubeola. Catarrh. Bron. Cat. Erysip. Pleurisy.	2 8 3 2 4 4 5	2 8 3 2 4 4 5	Ind. —	0	Amenorrhœa; hystero- epileptic fits, 2 y., robust.	Feverish- ness, out of sorts.	r.	L. base.	Sev. 11 d. Grad.	12 d.	R.	0	
1024 "	M.	49	Temp. Suff.	Pneum. Scarlat. Catarrh. Bron. Cat. Erysip. Pleurisy.	3 10 4 3 1 1 3	3 10 4 3 1 1 3	Good. Good.	0	0	Catarrh.	r.	L. whole	Mod. 12 d. Grad.	14 d.	R.	0	
1025 T. Corbett, M.R.C.S.	F.	44	Temp. Suff.	Pneum.	2	2	Ind. Bad.	0	0	Catarrh.	r.	R. base.	Mod. 4 d. Grad.	8 d.	R.	—	2
1026 "	M.	30	Temp. Suff.	"	"	"	"	0	0	Catarrh.	r.	R. base.	Mild 5 d. Grad.	5 d.	R.	0	3
1027 "	F.	7	Temp. Suff.	"	"	"	"	0	Pneum. 4 y. ago.	Vomiting.	0	R. base.	Mod. 5 d. Sud.	14 d.	R.	Cough.	4
1028 "	F.	8	Tot. abs. Suff.	Pneum. Catarrh.	1 2	1 2	Good. Good.	0	0	Catarrh 3 d.	0	R. base.	Mod. 5 d. Grad.	10 d.	R.	0	
1029 "	F.	14	Tot. abs. Suff.	"	"	"	"	0	Typhilitis 3 y. ago.	Catarrh, cough, pain in side	r.	R. apex.	Sev. 5 d. Grad.	10 d.	R.	0	5

F.R.C.S.	Sex.	Temp.	Tonsil.	1	2	Good.	Cough.	Enterie 3 y. ago.	Malaise, headache, cough.	r.	L. base.	8 d. Grad.
1031	M.	32	Temp. Suff.	1	1	Good. Ind.	F. } bronch. B. }				21 d.	R. 0
1032 T. F. Pearce, M.D.	F.	46	Temp. Suff.	1	1	Ind. Ind.	0	Syph. disease of cord and brain.	Sciatica, sickness.	0	Central sternal	Mod. 8 d. Sud. 6
1033 John E. Garner, M.D.	M.	18	Temp. Suff.			Good. Good.	0	0	Pain in side.	r.	R. base. L. apex.	Mod. 7 d. Grad. 0
1034 A. Creswell Rich, M.B.	M.	34	Tot. abs. Suff.	2	4	Good. Ind.	F. d. bronch. S. chr. bron. and emphys.	0	Malaise.	r	R. base.	Sev. 10 d. Grad. 0
1035 M. Messiter, M.R.C.S.	M.	15	Tot. abs. Suff.	2	+	Ind. Bad.	0	0	Eareche, headache.	r.	L. base.	Sev. 6 d. Sud. 7
1036	M.	16	Temp. Suff.	5	+	Bad. Bad.	0	0	Vomiting, headache.	0	R. base, then R. apex.	Mod. 12 d. Grad. 0
1037	M.	19	Intemp. Suff.	5	+	Bad. Bad.	0	0	Pain in side.	r.	R. whole	Mod. 13 d. Sud. 0
1038 T. E. Underhill, M.B.	M.	41	Intemp. Suff.	+	2	Ind. Ind.	0	0	—	r.	Both bases.	Sev. 10 d. Grad. D. 10th d
1039 Hugh R. Ker, F.R.C.S.	F.	33	Temp. Suff.	+	+	Bad. Bad.	0	0	—	r.	L. base. and apex.	Sev. 8 d. Grad. 0
1040	F.	13	Temp. Suff.	2	+	Ind. Bad.	M. pneum.	Strumous ophthalmia 12 m. ago.	—	r.	R. base	Sev. 8 d. Grad. 0

1. Roseolar rash, becoming papular in 2 d. over chest and limbs. 2. Cases, 1025, 1026, 1027 in same house; drain opened and left open close to house just before outbreak; case 1025, T. fell within 12 h. from 103.5° to 101.2° and remained so for 3 d., then fell to normal. 3. Case 1026, T. fell within 12 h. from 103° to 100° and remained so for 4 d., then fell to normal. 4. Case 1027, morning T. subnormal evening T. over 102° for 5 d. 5. T. varied from 101° in morning to 105° in evening; tongue dry, delirium, no control over sphincters for 2 d.; these two cases in another house, drain opened close by a few d. before attack. 6. Passed coffee coloured urine twice in 3 d. 7. Deafness during attack; no aural disch. 8. Always a heavy drinker; began with sev. pain in R. side of diaphragm, and R. plicurisy.



Observer's Name.	Sex.	Age.	Temperate or otherwise. Food.	Concurrent cases of illness in	Same house.	Same district.	Sanitary state of house " " of district	Family history of lung disease.	Previous illnesses of patient	Premontory symptoms.	Mode of onset.	Part of lungs affected.	Character of fever Duration Termination	Duration of physical signs.	Result.	Sequelae.	Note.
1041 E. G. A. Walker, M.R.C.S.	M.	14	Temp. Suff.	Pneum. Bron. Cat.	1	1	Ind. Good.	B. pneum. 2 y. ago. M. asthmatic.	Pertussis 12 y. ago, measles 11 y. ago.	Catarrh.	r.	L. base.	Sev. 7 d. Sud.	17 d.	R.	0	
1042 Herbert S. Renshaw, M.D.	M.	14	Tot. abs. Suff.	Tonsill. Herpes.	1	1	Good. Good.	2 S. } pneum. 1 B. } F.'s B. d. phth.	<i>Paenem.</i> 6 y. ago Diphth. 8 y. ago " 3 y. ago	Cold extrem.	r.	Both bases.	Sev. ? 12 d. Grad.	? 12 d.	D.		
1043 T. W. Gooch, M.R.C.S.	M.	28	Temp. Suff.	Pneum.	1	1	Good. Good.	0	0	Pain in L. side and abdom.	r.	L. base.	Sev. 5 d. Sud.	16 d.	R.	0	1
1044 "	M.	17	Temp. Suff.	Pneum.	1		Good. Good.	Many rela- tions phthi- sical.	0	Faintness, bilious vomg., pain in R. side.	r.	R. base.	Sev. 5 d. Sud.	—	—	Consid. pleural effusion.	2
1045 G. R. Allan, M.D.	M.	29	Intemp. Suff.	Pneum. Measles. Tonsill. Bron. Cat. Erysip.	4	6	Bad. Good.	0	0	Headache, anorexia.	r.	R. base.	Sev. 7 d. Sud.	—	R.	0	3
1046 Laurence Humphry, M.B.	M.	19	Nearly tot. abs. Suff.	Catarrh. Tonsill. Bron. Cat.	1	2	Good. Good.	0	0	Langnor 6 w. anorexia.	r.	L. base.	Mod. 6 d. Sud.	22 d.	R.	0	4
1047 R. Ross, L.R.C.P.	M.	38	Temp. Suff.	0			Good. Ind.	0	0	Pain in front and back of chest, dyspnoea, sickness.	r.	Both bases.	Sev. 7 d. Sud.	7 d.	D. 7th d.		5
1048 E. Barnes, M.D.	M.	63	Temp. Suff.	Pneum. Enteric. Catarrh. Tonsill. Bron. Cat.	1	4	Good. Ind.	0	0	Cough, pain in side.	0	R. 1. apex. 2. base. L. 3. apex.	Sev. 12 d. Grad.	about 21 d.	R.	0	6



Observer's Name.	Sex.	Age.	Temperature or otherwise.	Concurrent cases of illness in	Same house.	Same district.	Sanitary state of house " " of district	Family history of lung disease.	Previous illnesses of patient.	Premontory symptoms.	Mode of onset.	Part of lungs affected.	Character of fever { Duration Termination }	Duration of physical signs.	Result.	Sequelæ.	Note.
1059 W. J. Mickle, M.D.	M.	32	Temp. Suff.	Pneum. Bron. Cat.	2	2	Good. Good.	—	Diabetes insip. 10 y. ago, boils 2 y. ago and 1 y. ago.	Vomiting, anorexia.	r.	R. lower lobe.	Mod. 11 d. Grad.	16 d.	R.	0	
1060 John Reid, M.B.	F.	17 m.	Tot. abs. Suff.	{ Rötheln. Measles. Scarlat. Bron. Cat. Erysip.	5	3	Ind. Good.	—	—	Bronch.	—	R. apex.	Mod. till D.	till D.	D.		
1061 "	F.	30	Temp. Suff.	Pneum. Crop. Tonsill. Bron. Cat. Erysip.	1	1	Ind. Good.	—	—	Tonsillitis, constipation, gen. mala. se.	0	R. base. L. whole	Sev. 9 d. Grad.	9 d.	R.	Anæmia.	1
1062 John Griffin, M.B.	M.	41	Temp. Suff.	0			Good. Good.	0	Ague 24 y. ago and 17 y. ago.	Headache, coryza, pain in chest.	0	R. apex. L. base. & apex.	Mod. 21 d. Grad.	24 d.	R.	0	
1063 A. W. Leachman, M.D.	F.	14	Temp. Suff.	0			Ind. Ind.	—	—	0	r.	R. apex.	— 7 d. Sud.	—	R.	0	2
1064 "	M.	62	Temp. Suff.	0			Ind. Ind.	—	Erysip. of leg 9 y. ago.	—	r.	R. base.	Mod. — Sud.	—	R.	0	3
1065 W. F. Cleveland, M.D.	F.	48	Temp. Suff.	0			Good. Good.	—	Subject to bron. cat. and chr. eczema.	Hepatic congestion with sickness 2 d. prov.	r.	L. lower lobe.	Sev. 10 d. Grad.	Dulness 8 w.	R.	0	4
1066 "	M.	27	Temp. Suff.	Scarlat. Catarrh. Bron. Cat. Erysip.	3	3	Good. Good.	0	Enteric 3 y. ago.	Headache, thirst, pain in side.	r.	R. base.	Sev. 12 d. Grad.	17 d.	R.	0	5

## Original Communications.

[\*\* The Committee is not responsible for the statements or opinions contained in the papers under this heading.]

### AN EPIDEMIC OF PNEUMONIA IN THE PUNJAB.

BY SURGEON-MAJOR S. E. MAUNSELL.

*Army Medical Department.*

ANALYSIS of 85 cases of pneumonia occurring at Mooltan, Punjab, between 1st July, 1882, and 28th February, 1883.

Average strength for the period	702	
Total number of cases	85	} Including 9 camp followers.
Total deaths	43	

#### I.

Table showing admissions to hospital from the principal diseases during the above period.

Months.	Ague.	Diarrhoea.	Dysentery.	Pleuritis.	Bronchitis.	Pneumonia.	Rheumatism.	Skin Diseases.	Total.
July 1882.	13				1				14
August	253	1	3	3		2	1	2	265
September	205	5	8						218
October	279	1	18		2			1	301
November	89	2	5		20	13	1	1	131
December	33		2		40	24			99
January 1883.	19	1	3		38	34			95
February	20		1		20	3			44
November 1882.	<i>Camp Followers.</i>					2			2
December						7			7
Total.	911	10	40	30	121	85	2	4	1176

Average age of men attacked with pneumonia 25·09 years.

Average service do. do. 10·16 years.



## II.

Table showing the ages of those who died in quinquennial periods, with the percentage of deaths at each age.

Ages.	Total No. of deaths.	Percentage of deaths to ages
15 years and under.	3	6.97
16 to 20 years.	10	23.25
21 to 25 years.	17	39.53
26 to 30 years.	5	11.62
31 to 35 years.	2	4.65
36 to 40 years.	4	9.30
41 to 45 years.	1	2.32
46 years.	1	2.32

From this table it would appear that the mortality was greatest for the ages 21 to 25, and then from 16 to 20 years. From 16 to 25 the mortality was very high; before 16 and after 25 it was much less.

## III.

Table showing the deaths, with their percentages at different services, from 6 months to 21 years (9 camp followers unenlisted, not included).

Service.	No. of deaths.	Percentage to service.
Recruits of 6 months and under.	4	11.42
From 6 months to 1 year.	11	31.42
„ 1 year to 2 years.	3	8.57
„ 2 years to 3 years.	2	5.71
„ 3 years to 5 years.	4	11.42
„ 5 years to 10 years.	5	14.28
„ 10 years to 15 years.	3	8.57
„ 15 years to 20 years.	1	2.85
20 to 21 years.	2	5.71

The foregoing table shows that over 31 per cent. of deaths took place amongst soldiers of from 6 months' to 1 year's service; from 5 years' service to 10, the percentage was 14·28, or less than half; that recruits under 6 months' service, and men whose service ranged between 3 and 5 years suffered alike:—while the mortality was least among men of between 15 and 20 years service.

## IV.

Table showing religion and caste in cases attacked by pneumonia with mortality.

Religion.	Caste.	Average strength.	Average age.	Average service.	Attacked.	Percentage to strength.	Percentage of deaths to attacks.	Habits of life.
Mahomedan.	Mussulman.	210	21·4	8·67	18	8·57	38·88	} Eat meat, do not drink.
	Pathan.	100	24·	10·85	4	4·	50·	
Hindoo.	Sikh.	398	26·36	11·23	46	11·5	45·65	} Drinks spirits. Does not drink spirits. } Meat eaters.
	Dogra.	88	27·27	8·27	8	9·	62·50	

From this table it appears that the Mahomedans suffered less than the Hindoos,—7 per cent. of the former had pneumonia, and over 40 per cent. died; compared with 11 per cent. of the latter attacked, and 54 per cent. of deaths. It also seems from the notes as to habits of life, that the Mahomedans do not drink spirits, although free meat eaters; while the Hindoo eats meat and generally drinks spirits.

## V.

Table showing monthly percentages of admissions from four principal causes, to total admissions, during the eight months July, 1882, to February, 1883.

Months.	Ague.	Pneumonia.	Bronchitis.	Diarrhoea and Dysentery.	Percentage of Respiratory diseases to total admissions.
July 1882.	61·90				
August	88·15	·69		1·39	·69
September	62·12			3·93	
October	85·32		·60	5·81	·60
November	63·12	9·21	14·18	4·96	23·29
December	29·27	22·32	34·82	1·78	57·14
January 1883.	17·39	32·11	33·93	3·66	66·04
February	32·25	4·83	32·25	1·61	37·08

In this table we note that in the month of July, admissions from ague, which had evidently been prevailing in the regiment for some time, form nearly 62 per cent. of the total admissions for that month. The climax of the admissions for this disease takes place in the month of August when it rises to over 88 per cent. This is in keeping with the ordinary course of intermittent fever in the Punjab; the greater number of admissions take place in the month following the rain and not during the actual rainfall. On the 19th August out of 21 admissions to hospital, 18 alone were for ague; about this date also the first admission occurs for pneumonia: the curve of admissions for ague then descends in an irregular manner to the end of February, 1883, where the further medical history of this affection in the regiment stops short, with the rate of admissions more than 32 per cent. Pneumonia disappeared until November.

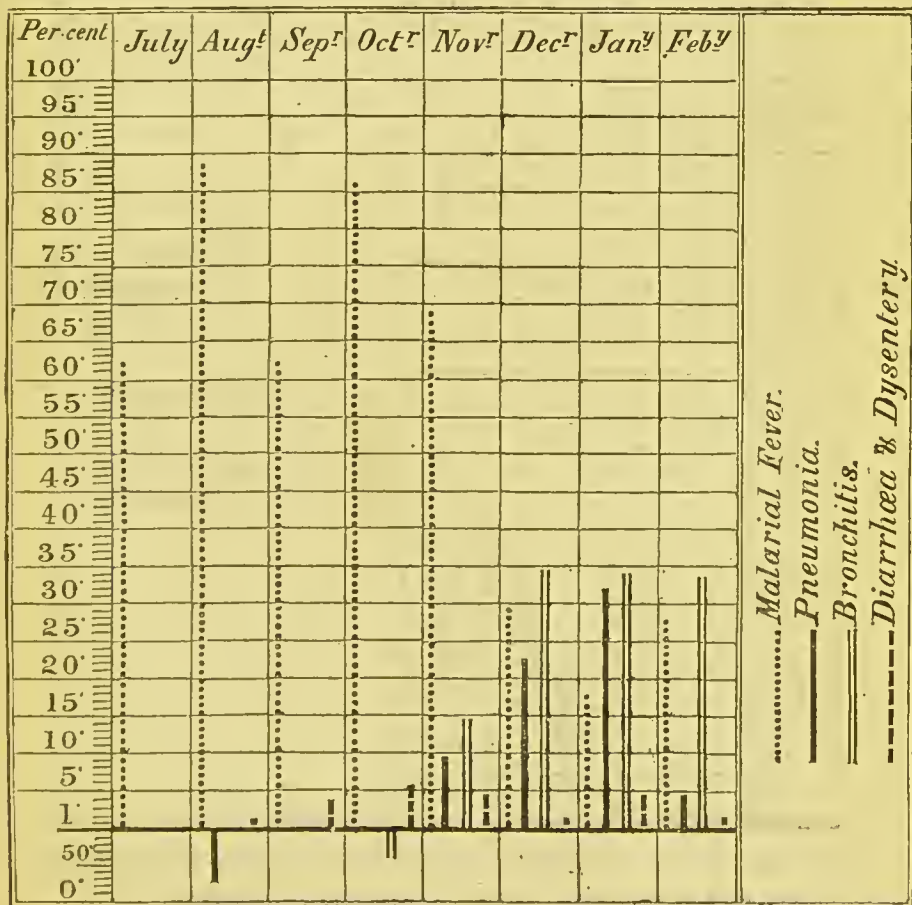
Next we find bronchitis appearing about the middle of October, forming two admissions out of a total of 327 for that month. In the following month of November we find the admissions from this disease rising to over 14 per cent., and in

December to over 34 per cent: when the curve falls gradually, breaking off at 32.25 per cent. in February: the report does not carry us further than this month.

Pneumonia follows next, leading off in the month of November with 13 admissions out of 141, or over 9 per cent.: following the curve of bronchitis it then rises in the month of January to over 32 per cent.; finally disappearing from the field of view in the month of February, where in that month the admissions from this disease form under 5 per cent.; the bronchitic curve still proceeding. The curve traced by each of these diseases is shown in the accompanying diagrams A. and B.

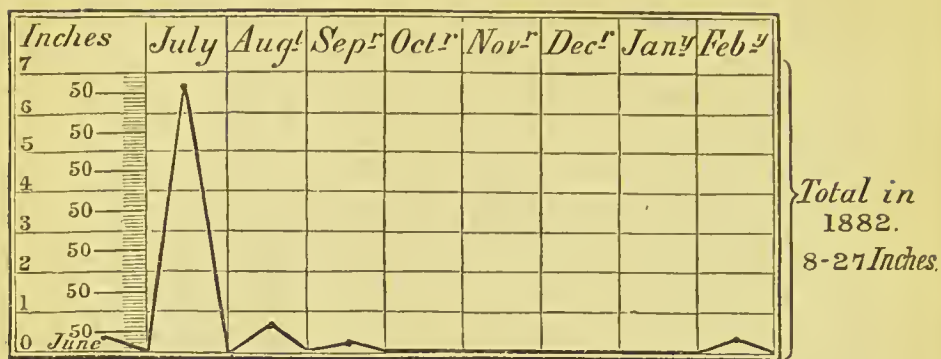
## A.

Diagram of Percentages of Admissions to Hospital for the four principal diseases, between 1st July, 1882 and 28th February, 1883.



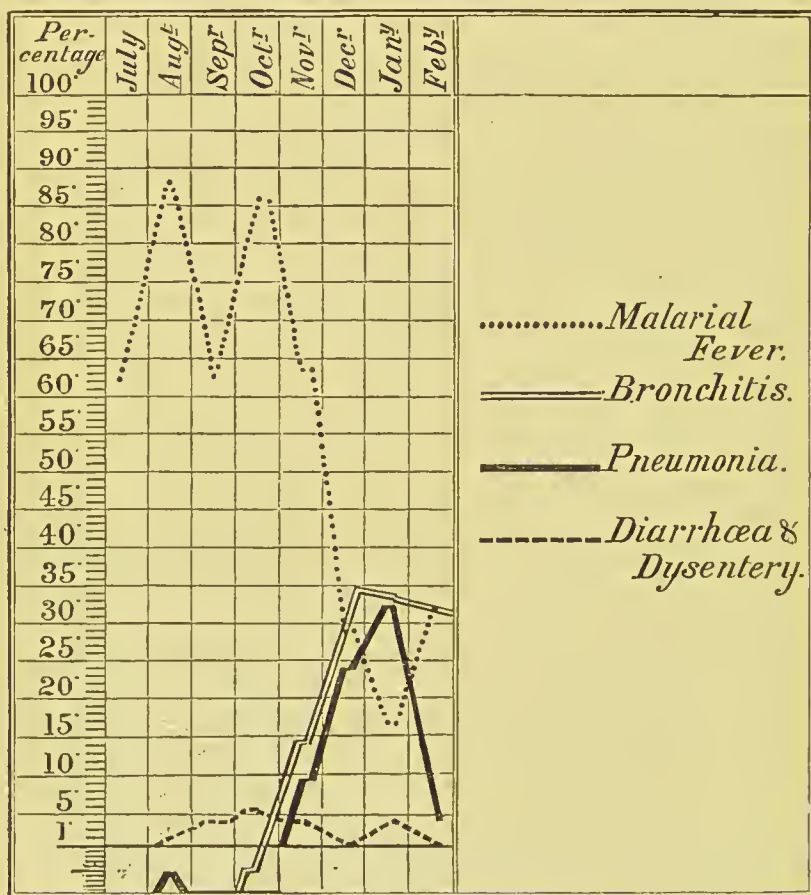


## Rain Fall.



## B.

Diagram of Percentages of Admissions to Hospital for the four principal diseases, between 1st July, 1882 and 28th February, 1883.



The last column in Table V. shows the ratio of admissions from these two diseases combined, to total admissions.

Of the 85 cases attacked by pneumonia 42 recovered—the average duration of each case was under 24 days—20 had suffered once only during the previous twelve months from ague, and the average duration was 4·05 days: the average interval which elapsed before the pneumonic attack was 61·05 days.

Seven suffered from bronchitis during the previous year—in 3 cases this affection was of 11 days' duration—the average intervening interval before the pneumonic attack was 30 days.

In 4 cases bronchitis was a complication of the pneumonia.

The remaining 15 cases had not been under treatment during the previous twelve months.

Of the 43 fatal cases—

The shortest terminated fatally in 12 hours.

4 cases                   "                   "                   2 days.

4 cases                   "                   "                   3 days.

The average duration of each case was 6·65 days, and the longest was 18 days.

Nineteen of these suffered from one attack each of ague during the preceding twelve months, the average duration of each case was 7·7 days, and the average interval before the pneumonic attack was 61 days.

One had suffered from dysentery for 9 days.

Five suffered from bronchitis as complications of the pneumonia

One from bronchitis at an earlier period of the year.

One had been under treatment for splenitis 109 days, and was attacked by pneumonia while in hospital,

Sixteen had not been under treatment during the previous twelve months.

Out of the 85 cases, therefore, 39 had had ague once during the previous twelve months, the average duration in each instance was between 5 and 6 days, and the average interval which elapsed before the pneumonic illness was 61 days.

It cannot be considered that these slight attacks of intermittent fever, occurring in men who are shown to have had freedom from other affections during the preceding twelve months, could have had much, if they had any, influence, either in predisposing to, or in exciting the subsequent pneumonia;

which in the average did not make its appearance until 2 months subsequently.

From the report it seems that out of 911 cases of ague the various castes were affected in the following sequence:—

Sikhs.

Mussulmans.

Pathans.

Dogras.

And on referring to Table IV. above, it will be seen that they were in the same order of sequence subsequently attacked by pneumonia. This, however, is only a coincidence from which no fact can be adduced to support a supposition that the diseases were connected; as it has been shown that the previous prevalence of ague influenced very little, if at all, the subsequent outbreak and course of the respiratory affection; and Table IV. further shows that of the two principal classes into which these four castes resolve themselves, and in which they become fused, viz., Mahomedan and Hindoo, the latter suffered from a higher percentage of mortality than the former. The Hindoo Dogras, who do not drink spirits, died off in greater numbers than the Sikh, who were attacked in greater numbers, and who do drink spirits: also that the Mahomedan Pathan, who is not a spirit drinker, suffered more than the Hindoo Sikh: that of the four castes the spirit drinkers stood 3 down the list in rate of mortality.

Short notes are given of 5 cases which terminated fatally, and all the 85 were apparently marked by very similar symptoms.

Two cases were admitted in a state of exhaustion, and died the same day: this sudden termination points more, I think, to pulmonary collapse than to ordinary fatal cases of acute pneumonia.

One case was admitted with a temperature of  $102^{\circ}$  rising rapidly to  $104^{\circ}$  F., oscillating between  $101^{\circ}$  and  $103^{\circ}$  F., till he died on tenth day, temp.  $104^{\circ}$  F. His pulse during that time varied from 108, 126, 137, 150, the respirations 25, 35, 48, 50. On admission his cough was marked by severe pain, he had great difficulty of breathing, in fact orthopnoea, bubbling râles are heard over the posterior region of left lung inferiorly, cough short and choking, large quantities of expectoration containing bright

red blood, anxious countenance and early delirium, dulness on percussion over both bases posteriorly. On the same day he became delirious, pulse 144, skin wet with perspiration, became unconscious and seemed about to die. He suddenly improved, and the following day was much better, the bases of both lungs remain dull, bubbling râles are heard over the surface, can lie comfortably, and the urgency of the symptoms abate; but he continues in a very weak and prostrate state, coughs up large quantities of purulent expectoration till the tenth day, when the case terminates fatally.

I think it may be concluded that this was a case which commenced with catarrhal bronchitis, and became subsequently complicated by catarrhal pneumonia: this is testified to by the painful nature of the cough, the dyspnoea, or orthopnoea, and the rapidly increasing elevation of temperature; also the very quick ratio of the respiration and pulse. Death seems to have been threatened on the first day from pulmonary collapse.

Second case, admitted with temperature 102° F., cough frequent, severe, and very painful, respiration 40, pulse 120, both lungs dull posteriorly, large râles audible in both, great exhaustion; temp. rises to 105°, and death the same day.

Third case admitted with fever, temp. 102° F.; his expectoration soon shows blood, which is sometimes absent for two days at a time; dulness is found in the lung substance over lower  $\frac{2}{3}$  posteriorly, and "very little respiratory action"; large bubbling râles are heard. He finally recovers after a protracted illness of fifty days, attended with great prostration.

Fourth case admitted in an exhausted state with cough and fever; died on third day. No blood appeared in his expectoration.

Fifth case admitted in an exhausted condition, and died on same day.

Attached are three temperature charts:—

a. Chart of case No. 1 above.

b. Typical chart of acute catarrhal pneumonia.

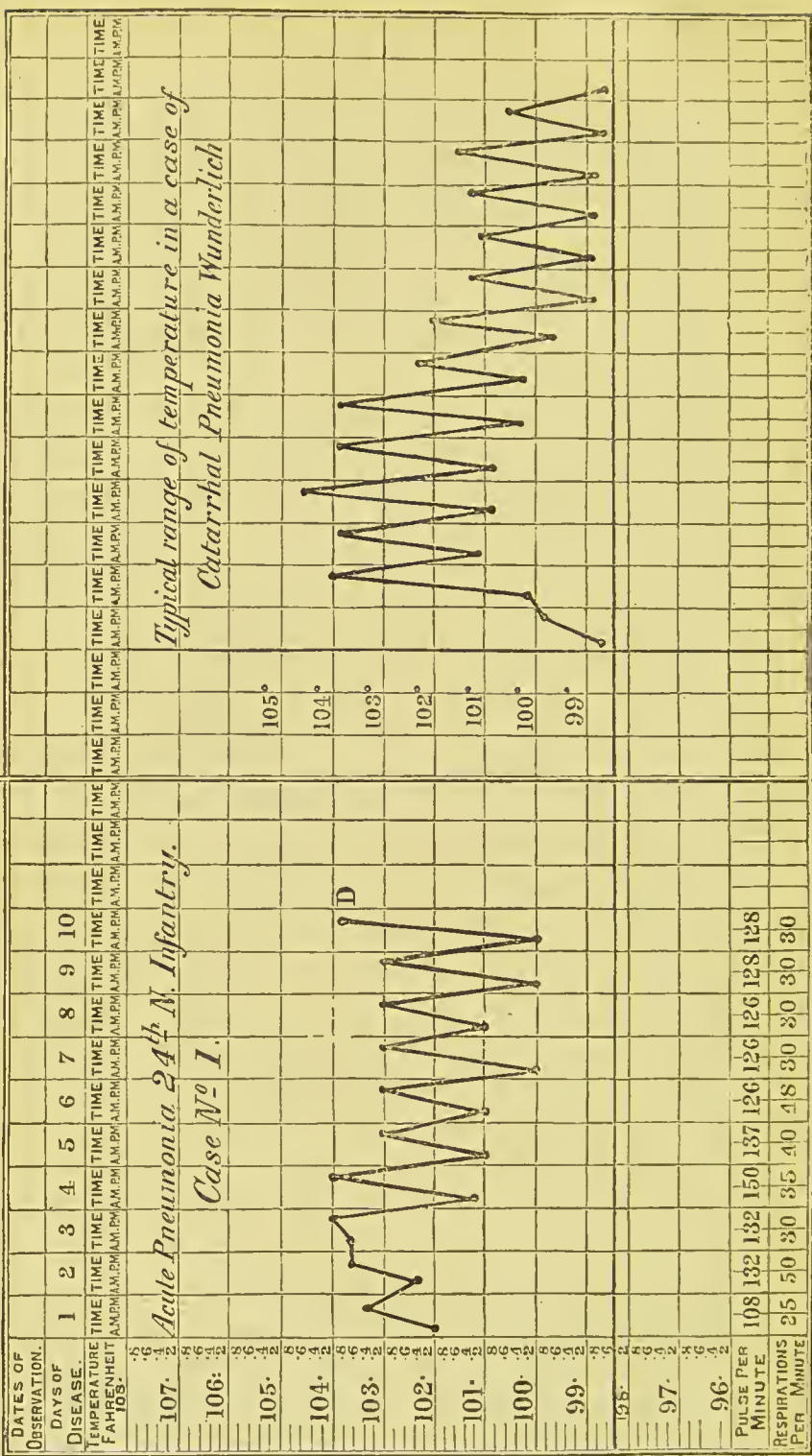
. Typical chart of acute pneumonia (croupous).



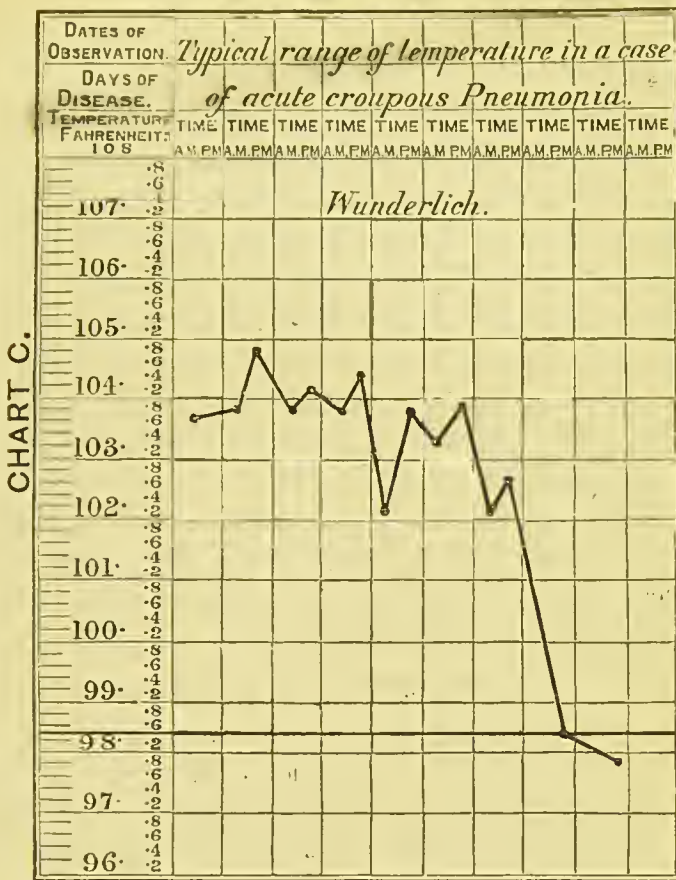
CHART A.

CHART B.

Age 42 Years. Service 22 yrs. Name. H.B.S.  
Result C.



*Disease, Pneumonia. Date of attack*



All the 85 were apparently marked by the same or very similar symptoms, which may be summarized as follows:—

A pre-existing feverish cold, followed by a markedly painful cough, urgent dyspnoea or orthopnoea, rapidly ascending temperature, with a similar very high ratio of pulse and respirations; the bilateral condensation of the lungs, the symmetrical subcrepitant large bubbling râles, extreme anxiety of countenance and prostration, the complete absence or very marked weakness of respiration sounds, the very variable character of the expectoration—free rather than scanty, mucus or purulent or florid red rather than tenacious or prune-juice-like—and the sudden termination, all point to acute capillary bronchitis, tubes blocked by copious secretion, with condensation and collapse of lung as a result, or possibly catarrhal bronchitis with catarrhal pneumonia.

"Initial rigor" is not noted in the cases given, and is presumed to have been absent.

The diagram B. shows that the original disease was bronchitis, the so-called pneumonia appearing as a complication. These two diseases then proceed conterminously, bronchitis keeping always a-head, and pneumonia forming a complication in 27·27 per cent. They both reach the summit of their curve, bronchitis 34 per cent. in December, pneumonia 32 per cent. in January, and then both decline, the latter during February, the former still continuing at the rate of 32 per cent., and doubtless in its turn disappearing in following month. It would therefore seem that pneumonia was a complication added to a previously and extensively existing bronchitis, and that the epidemic was not one of pneumonia alone.

American authors believe that acute catarrh of the smaller bronchi is more common in adults of both sexes than is generally supposed, and that it was not uncommon among the American soldiers during the American war, especially the coloured troops, and that it was often mistaken for pneumonia or ordinary acute bronchitis.

The condensation of the lung which is alluded to was most probably that which often accompanies acute bronchitis (capillary). Professor Aitken, Dr. West, and Dr. Gairdner describe the complication as follows:—

"One most direct, invariable, and important result of bronchitis in the adult is condensation of the vesicular substance of the lung of a peculiar type, as a result of mucous or other obstruction in the air-tubes leading to the condensed portion.

"Such condensation is produced by pulmonary collapse; when extensive and sudden is not only frequently a cause of death, but at the same time it is a fleeting, temporary condition of immense frequency, and important practically to distinguish from the condensation of pneumonia. . . . Such collapse and condensation of the lung, however, whether lobular or diffused, is an exceedingly common lesion in the adult. . . . Is an almost invariable concomitant of fatal bronchitis—depends on obstruction of the tubes."

Without the light which post-mortem examinations would shed on these cases, it cannot be said to what extent acute



uncomplicated pneumonia prevailed, and to what extent it existed as a complication of bronchitis; but judging by the symptoms quoted and from the medical history of the corps at the time—seeing that bronchitis had gained a footing of 14·18 per cent.—it is only reasonable to suppose that the majority of the 85 had catarrhal or capillary bronchitis to commence with, and, probably owing to neglect on the part of the individual, or from exposure to variations of temperature, or to cold winds which prevail during the winter at Mooltan, catarrhal pneumonia as a complication, some of them dying rapidly with symptoms of pulmonary collapse.

In endeavouring to assign a cause for the unusual prevalence of bronchitis and pneumonia at Mooltan at the time, we must look to the natural history of those diseases and to local and climatic influences, especially rapid and great variations of temperature, as the sources from which information may mostly be expected.

Pneumonia often appears in the epidemic form in this country, Europe, and America; also epidemic catarrh or influenza—in Europe not only amongst the population, civil and military; but as an epizootic amongst animals—and is stated to be a disease of extraordinary rapidity of progress and great diffusibility, and if the epidemic influence is great, it is frequently accompanied by dyspnoea, great debility, profuse perspirations, capillary bronchitis, and pneumonia.

Could the outbreaks of bronchitis or pneumonia which occasionally occur in this country be traced to the epidemic influence which gives rise to influenza, a very interesting point in the natural history of that affection would be settled, as it appears to be not very common in India.

Professor Aitken says that in 1557 an epidemic of influenza had its origin traced back to its starting-point in Asia, whence it proceeded westward to America. Also, occasionally it has originated in India, but more commonly in Northern Europe—Moscow, Warsaw, Dresden. Again, a catarrhal fever is known to occur in India, occasionally spreading along the banks of some river and subsiding again.

Originating generally in the East in epidemic form, its course is marked by a rapid progress in a westerly direction; and in Europe it usually assumes the endemic character.



As regards pneumonia, also—putting aside the forms which are known to exist as complications of enteric or typhus fever—it is of not unfrequent occurrence in a form on which endemic or epidemic influence has impressed a peculiar character; and in America malarial regions exhibit this in what is known as the “winter fever,” a typhoid pneumonia of the Southern States. It is marked by early and great debility, out of all proportion to the local symptoms. In Europe it sometimes appears as if it were an epidemic. Aitken says that in 1862–63, in America, it caused a mortality of from 26·82, 24·14 per cent. in the Confederate Army, where it was, in the opinion of Dr. Clymer, in many cases confounded with capillary bronchitis.

Now, coming to the Punjab, it appears that respiratory diseases always form a high percentage of admissions,—for instance in the periods from 1867 to 1876 respiratory diseases show an average admission rate of 52· per 1000 of strength amongst native troops (according to Bryden’s “Vital Statistics of India”),—and that 33 per cent. of deaths from all causes were due to diseases of the respiratory organs; that the greatest mortality was generally in November, December, January, and to the end of February, presumably owing to the wide variations of temperature.

Great diurnal range of temperature is a marked feature in the Punjab, and in some places, notably at Mooltan, the absolute range may amount in the winter months to 47° or 50°. The annual extremes are very great at Mooltan or Dera Ismail Khan, the temperature in the shade ranges from 29° to 126°, and the Punjab at opposite seasons of the year, mainly on account of its great seasonal variation of temperature, is the seat of the highest and lowest variation of pressure, due allowance being made for altitude in Northern India.

Another marked feature of this climate is its dryness; thus, while the mean humidity of Calcutta is 76, and of Allahabad 53, that of Lahore is 44·5, and of Mooltan 43. The dryness of the soil again compares with that of the atmosphere; only very rarely do we find the ground presenting impenetrable strata to arrest the descent of water, while the rainfall over the greater part of the surface is scanty and the rivers are widely separated.

The chief mortality amongst the native troops, excluding

epidemics, is from respiratory diseases, fevers, dysentery and diarrhœa.

As regards the immediate district of Mooltan itself, it differs in no respect in physical features from the Punjab generally. The water is found at a depth of about 25 feet from the surface, the depth varying little at the different seasons, and even in the neighbourhood of the large rivers—the Sutlej and the Chenab—is apparently little affected by their rise and fall.

The climate is very dry ; in October or November the heat of the sun is moderate, while the nights are cold, and at sunset a cold cutting wind from the North frequently springs up. The rainfall averages 7·19 inches per year ; some years the fall does not reach 2 inches ; in 1882, it was 8·27 inches, the greater part of which fell in July.

Diseases of the respiratory organs are common amongst the troops during the cold season, and as a rule form the chief cause of mortality.

Amongst the European troops in the 10 year period, 1860 to 1869, the average annual admission rate per 1000 was 86. The most commonly fatal disease of the class is pneumonia. About the cause of the disease there is considerable difference of opinion, but that which receives most general support is that amongst the native troops the disease, though it has frequently an epidemic character in a station or regiment, is not contagious, and that it is due to cold impressing itself upon the men either through the skin or bronchial tubes whilst on night duty, or even still more commonly when off duty, owing to the need of sufficiently protective undress clothing.

In the epidemic under review no evidence has been adduced which would favour the idea that the disease was communicated by contagion or infection.

In the jails in this province respiratory diseases are common and very fatal ; in certain jails outbreaks of pneumonic typhus have occurred during typhus periods, and have been associated with contagious diseases such as diphtheria and erysipelas, suggesting that the pneumonia was also due to contagion ; but nothing seems to have been clearly proved.

In 1875, pneumonia was very prevalent and very fatal on the Punjab frontier ; at the same time an outbreak of that disease

occurred at Mooltan. The civil surgeon reporting on the subject of the pneumonia on this occasion says :

“There is nothing in the meteorological phenomena of the year to account for the fact (the prevalency of lung diseases amongst the prisoners), for the present cold season has not been more severe than, if it has been as severe as, the previous one ; and so far as I can discover the prisoners have been subject to no new conditions which can have favoured the production of pneumonia.

“The theory that pneumonia or pleuro-pneumonia is infectious would furnish an easy explanation of the facts of the case. But then, is it infectious? I must state that so far as I can judge the past year's experience of the disease in the Mooltan jail affords no proof of that doctrine.”

The accumulated experience of the 8 years which have since elapsed leaves the matter still in the same position.

## REMARKS ON A SECOND EPIDEMIC OF PNEUMONIA OCCURRING IN THE PUNJAB, BENGAL, 1882-83.

BY SURGEON-MAJOR S. E. MAUNSELL.

*Army Medical Department.*

SINCE sending you a report on an epidemic of Pneumonia occurring in the Punjab, I have had an opportunity, through the kindness of the Surgeon-General, H.M.'s Forces in India, of reviewing another similar epidemic which has occurred recently at the station of Edwardesabad or Bunnoo, in the Trans-Indus district of the Punjab frontier of Afghanistan.

Edwardesabad is a small military cantonment 1276 feet above the sea level, lat.  $33^{\circ}$  North., long.  $70^{\circ} 39'$  East; the average rainfall is 11 inches; the water is fairly good, supplied from wells which are of great depth. It is said that the men and the children are liable to suffer from fever and enlarged spleen, as well as diarrhœa; however, none of these affections seem to have been unusually prevalent during the course of this epidemic. Generally speaking the rate per 1000 of admissions for respiratory diseases amounts to a little above 71. The greatest number of admissions in the year are as a rule caused by intermittent fever, next in order comes dysentery, then diarrhœa, respiratory diseases holding the third place. Average mean temperature for the year is about  $73^{\circ}$ ; in winter the station is liable to suffer from extremely cold winds; the contrast between the heat of the day and the cold at night is very great; the atmosphere contains much moisture, and during the later months of the year it is damp and cold.

The following table shows the two epidemics, the one I have already communicated, as well as the one which is the subject of this letter; compared as regards the season of prevalence, the rate of attack, and comparative mortality.



94 SECOND EPIDEMIC OF PNEUMONIA OCCURRING

No. 1.

Months.	1st Epidemic	2nd Epidemic.		Remarks.
		A	B	
January.	34	39	{ 89 cases occurred during 5 months of cold weather = 11· per cent. of strength. 24 died = 27 per cent of attacked.	A and B represent the sickness and mortality from this cause, in two corps of native troops occupying the same station (Edwardesabad) who were simultaneously attacked.
February.	3	19		
March.	—	3		
April.	—	—		
May.	—	—		
June.	—	—		
July.	—	—		
August.	2	—		
September.	—	—		
October.	—	—		
November.	13	4		
December.	14	30		

With 9 unenlisted camp followers.  
 85 cases = 12·10 per cent. of strength.  
 43 deaths = 50·58 per cent of attacked.

95 cases = 13·04 per cent of strength.  
 27 died = 28·42 per cent. of attacked.

No. 2.

Table showing the apparent predisposition, as affected by race, and habits of life.

Religion.	Caste.	1st Epidemic.	2nd Epidemic.		Habits of Life.
			A.	B.	
Mahomedan.	Mussulmān.	8·5	22·50	13·07	{ Meat-eaters, but do not drink spirits.
	Pathan.	4·	10·	11·19	
Hindoo.	Sikhs.	11·58	8·86	5·74	{ —drink spirit. Eat meat. —do not drink.
	Dogras.	9·	22·64	13·17	
Poorbeahs, or	Mixed race.	—	22·22	25·	Mixed.

## No. 3.

Table showing apparent racial effect on mortality.

Religion.	Caste.	1st Epidemic.	2nd Epidemic.		Remarks.
			A.	B.	
Mahomedan.	Mussulmāu.	38·82	98·23	30·	
	Pathan.	50·	9·09	20·	
Hindoo.	Sikhs.	45·65	35·29	50·	
	Dogras.	62·50	25·	0·00	
Poorbeahs, or	Mixed race.	—	18·75	30·	

## No. 4.

The foregoing two tables exhibited in another form, or the order of greatest number attacked of different races.

1st Epidemic.	2nd Epidemic.		Remarks.
	A.	B.	
Sikhs.	Dogras.	Poorbeahs.	The Pathans, a hardy race of men, in each of these three instances had a comparatively few number attacked; next to them the Sikhs; the other races changing about, occupying different positions in each case.
Dogras.	Mussulmān.	Dogras.	
Mussulmān.	Poorbeahs.	Mussulmān.	
Pathans.	Pathans.	Pathans.	
—	Sikhs.	Sikhs.	

## No. 5.

Table showing the highest number of deaths according to race.

1st Epidemic.	2nd Epidemic.		Remarks.
	A.	B.	
Dogras.	Mussulmāns.	Sikhs.	This table again shews a difference as regards the mortality,—the Pathans still holding a low place in the list.
Pathans.	Sikhs.	Poorbeahs.	
Sikhs.	Dogras.	Mussulmāns.	
Mussulmān.	Poorbeahs.	Pathans.	
	Pathans.	Dogras.	

The next table shows how the corps marked A in the second epidemic noted above, fared in two successive years; contrasting the attacks and mortality as affecting the different castes.

## No. 6.

Attacked.		Mortality.	
1881-2.	1882-3.	1881-2.	1882-3.
Poorbeahs.	Dogras.	Sikhs.	Mussulmāns.
Mussulmāns.	Mussulmāns.	Poorbeahs.	Sikhs.
Sikhs.	Poorbeahs.	Mussulmāns.	Dogras.
Pathans.	Pathans.	Dogras	Poorbeahs.
Dogras, escaped.	Sikhs.	Pathans	Pathans.
		} escaped.	

A comparison of the above tables, Nos. 2 to 6, shows that caste and habit of life does not seem to have influenced the disease much, if at all; the Pathans, a Mahommedan sect who eat meat, but do not drink spirit, generally hold a low place down the scale; it may be also noted about this sect that the frontier of India is their native country. The other four castes oscillate up and down, holding a different level in each

epidemic; and this applies in the above instances not only to epidemics of different years, but as occurring at different stations, to the same regiment as affected in two successive seasons, as well as to two regiments in the same locality, in what for the sake of argument may be called the same epidemic area involved in the same epidemic wave.

In the first epidemic both lungs appear to have been implicated in the majority of instances; in the second epidemic, in the cases marked A, the right lung was affected in 38, the left in 35, both in 22 of the cases; amongst the cases marked B, no information is given.

As concurrent affections, may be noticed in the second epidemic, and in the same community, bronchial catarrh, coryza, mumps; but there is no mention of any specific fever, tonsillitis, herpes, or erysipelas. The medical officers having charge of these different cases, note in their report the weather being damp, cold, and cloudy; may not these meteorological conditions, which doubtless produced the bronchial catarrh and coryza, have also produced the pneumonia?

These men occupied barracks, or what are called Native Infantry lines, the sanitary condition of which has not been objected to in the report.

Family history under the circumstances in connection with natives is not traceable, and unfortunately information is not given as to previous illnesses, precedent rigors, the part of lungs affected, the character of expectoration, or range of temperature; it is, however, stated that in the cases marked A,

the average duration was 29·29 days,

that 11 died in 5 days or under,

10 died in from 6 to 10 days,

3 died in from 11 to 20 days,

3 died in over 20 days.

When these figures are compared with those showing the average duration in the first epidemic, in which the average duration of the 43 fatal cases was but 6·65 days, a marked difference will be noticed; it will also be noted that in the first epidemic some of the cases terminated exceedingly suddenly, 2 of them having died in 12 hours.

It is stated that the 95 admissions which occurred in A,



second epidemic, took place from 8 companies of nearly equal strength and performing similar duties, and living under similar hygienic circumstances.

As to the manner in which some of these men with their attendants were attacked, the following facts may be noted :—

“A,” admitted on December 30th; he had three attendants during his illness, A<sub>1</sub>, A<sub>2</sub>, A<sub>3</sub>; one of them, A<sub>1</sub>, got pneumonia on January 2nd and died; A<sub>3</sub>, became attacked on January 8th; A<sub>2</sub>, became attacked on January 11th. None of the men who subsequently attended on A, nor any of those who attended upon A<sub>1</sub>, A<sub>2</sub>, A<sub>3</sub>, became affected.

“B,” was admitted on December 26th, and died on January 5th; he had two attendants during his illness, B<sub>1</sub>, B<sub>2</sub>. B<sub>1</sub>, got pneumonia on January 7th, and died on January 14th; B<sub>2</sub>, got pneumonia on January 8th, and died on January 18th. Two men waited upon B<sub>1</sub>, during his illness, neither of them suffered. Two men waited upon B<sub>2</sub>, who got pneumonia on the 8th; one was attacked on the 19th and died on 28th, the other escaped.

These six cases were, therefore, attacked on the 2nd, 9th, 12th, 12th, 13th and 15th day of their attendance; on the whole, 12·67 per cent. of the sick nursing attendants were attacked; this is attributed, by the officers in charge, to hard work and exposure at night. Against this it must be noted that hospital officials, such as a dresser, two hospital assistants, the cook, watercarriers, and sweepers, whose duties required them to be in the precincts of the hospital always, as well as frequently in the wards in connection with the sick, throughout the whole period of this epidemic, escaped.

In this same second epidemic, B, about 20 per cent. of the attendants became attacked; it is also noted that some men who were in the habit of going to visit their friends in hospital during their illness, were attacked subsequently by pneumonia. In one case a “kahar,”\* C, was attacked on 27th January. His attendant, C<sub>1</sub>, became attacked by pneumonia on February 4th. Another “kahar,” D, was attacked on March 18th; he got better, then had a relapse. His friend who visited him during his

\* A Kahar is a native “letter bearer,” who has as a rule no special duty to do inside a native hospital; their services are, however, impressed occasionally when epidemics are about, and they nurse their own comrades in sickness.

illness was attacked on April 10th. A man who attended on him subsequently, became attacked on April 14th. Of 14 "kahars" doing occasional duty about the hospital, six became attacked; of them two only actually nursed the sick. On the other hand it is noted that two hospital assistants and one dresser, who were engaged through the wards during the whole of the epidemic, escaped; and of eight others, including cooks, and water-carriers, and sweepers, who had also duties to perform in connection with the sick throughout the whole epidemic, only one was attacked.

Although such a number of persons in this community became simultaneously or successively attacked by pneumonia, nothing has been put forward to prove the presence of any septic influence; while in each of the epidemics it is shown that bronchial catarrh prevailed beforehand or concurrently, and also that the meteorological conditions of the stations were such as would lead to pneumonic complications occurring in persons liable to or predisposed to bronchial affections.

No mention is made of investigations having been carried out with a view to the discovery of bacilli, or micro-organisms, in the sputum or lung tissues after death, which at present, in England and in Europe, are the subject of inquiry and speculation in connection with the pathogenesis of pneumonia. I presume that had such organisms been found the fact would have been noted.

With reference to cholera in this country, it is known that the circumstances of similar local and meteorological influences, the fact of dwelling in a locality where such influences prevail, and the enervating effects of prolonged and constant nursing, is a very important element of danger to attendants in cases of that disease; the same rule probably applies in cases of epidemic pneumonia.

In the second epidemic it is noted that many of the cases were ushered in by violent vomiting and purging. I see that the same features marked an epidemic of pneumonia which occurred at Stoke, in England, in 1882.

SALAR, *August 4th*, 1883.

ABSTRACT REPORT OF A SERIES OF (UNPUBLISHED) CASES OF EPIDEMIC PNEUMONIA, OCCURRING AT GARELOCHHEAD, FIRTH OF CLYDE, IN MAY, 1879.

*Communicated by*

JAMES FINLAYSON, M.D., GLASGOW.

OF 13 inmates of the house, 3 children and 2 adults were affected. The house was in good condition, and had been occupied before, and has been occupied since, by various groups of children without any illness. The drainage was unusually good, and the water was tested during the time the illness occurred, and nothing wrong discovered. No similar cases had occurred in the village.

THE GRANDMOTHER, who had been staying with others in the house all the winter, took ill on May 5th, with feverish symptoms; distinct physical signs of pleuro-pneumonia soon supervened in the lower part of the left side. She was very seriously ill for a time: although her illness was tedious, she recovered in two months or thereabout.

THE ELDEST CHILD, a girl,  $5\frac{1}{2}$  years, had been poorly for a day or two. From April 30th she was supposed to be better, and was even out for a little on May 2nd, but was completely laid up on May 3rd, with high febrile symptoms, and signs of pneumonia in the right side soon appeared. A rapid crisis with a fall of temperature from  $104^{\circ}$  to  $96^{\circ}$  (or  $97^{\circ}$  in rectum) occurred during the night of May 6th. Gradual convalescence was established, and her health has since been good.

OTHER TWO CHILDREN, both younger, took ill on May 5th with high fever, great restlessness, frequent and very painful cough, but no distinct signs of pneumonia could be made out. The acute illness terminated in these two cases on May 9th and May 10th, quite suddenly.

*Note.*—In all these cases given above there was much complaint of earache, with deafness for a time, and in one of the



children, actual suppuration. The children were all more or less delirious; no sore throat or rash could be discovered. . The remaining child, a baby at the breast, escaped any illness.

THE MOTHER, *ætat* 31, who had gone down with her children on April 10th, brought them home to Glasgow on May 8th, while they were ill, but she was herself apparently in perfectly good health and able to nurse a young child at the breast, as well as to attend to those who were sick.

She took ill very suddenly on May 10th, with vomiting, shivering, delirium, headache, and pains in the back and chest. The temperature ranged from  $102^{\circ}$  to  $105^{\circ}$ . The respirations were only a little accelerated till the sixth day, when they became rapid. Physical signs were practically absent till May 16th, only some slight restriction of breath sounds before that; dulness under right clavicle was then discovered. The cough was painful from the first, causing pain in the right shoulder, but it was not a very prominent symptom; it was more marked at the very beginning and at the end of the illness than at its height. No expectoration till the fourth or fifth day, and then only mucous sputa; the amount increased a little, later in the case: no rusty expectoration from first to last. After the dulness was detected on May 16th it rapidly deepened; the breath sounds became feeble and almost suppressed, without any tubularity. Towards the end of the case diarrhœa and involuntary evacuations occurred; a slight jaundiced tint appeared; great labour in the breathing supervened, and the febrile flush became tinged with lividity, and death occurred on May 19th. [Details of treatment omitted.]

This patient also complained of earache, with a little deafness but no discharge. No sore throat, no coryza, and no rash appeared.

The examination made by Dr. Joseph Coats showed grey hepatization of the upper lobe of the right lung, strictly demarcated and very typical; fibrinous plugs in the bronchi; red hepatization and engorgement in all stages in the lower lobes. A thin coating of recent exudation on the surface of the right lung, with soft adhesions in upper part, but no chronic disease. Left lung with some œdema. No engorgement of Peyer's patches in lower part of ileum. Nothing wrong with liver. No pus in the ear.



MEMORANDUM ON THE INCIDENCE OF FATAL  
PNEUMONIA.

BY G. B. LONGSTAFF, M.A., M.B., OXON., M.R.C.P., F.S.S.

THE returns of the Registrar General's Department necessarily only deal with *fatal cases*. Bearing this in mind, the following table, based as it is on more than twenty-four thousand cases, has a value which admitted inaccuracies in the returns cannot destroy.

The figures are obtained from the Annual Reports for the several years, and from the Census of 1881, vol. iii., and show the following facts for males and females, separately:—

(1) The number of deaths from Pneumonia at all ages and at various groups of ages for each year from 1871 to 1880, both inclusive.

(2) Do. do. do. for the decade 1871–1880.

(3) The *average* number of deaths, &c., &c., for the decade.

(4) The *average* Death Rate per million living at each age: (mean of the two censuses, 1871 and 1881).

It should be noted that males suffer more than females, almost in the proportion of 3 : 2. But the disparity is slight at ages 2—15, and is most marked at ages 35—65, when males suffer more than females almost in the proportion of 2 : 1.

The greater mortality of male infants can only be dependent upon that difference in the male constitution which causes the well-known excessive mortality of males under 2 years of age. The greater mortality in middle life may conceivably be due to sexual differences, or more likely to occupations or drinking habits.

In both males and females, deaths from Pneumonia are proportionately least frequent at ages 10—15, increasing on either

side of that point to the extremes of life—but the mortality is three times as great, according to the return, in the first year of life, as in old age.

The facts are more clearly shown in the diagram, in which the black curve represents the male death rate, the red curve the female death rate. For years 3—15 they are practically coincident.

The winters 1870-1, 1874-5, 1878-9, 1879-80, 1880-1, were severe.

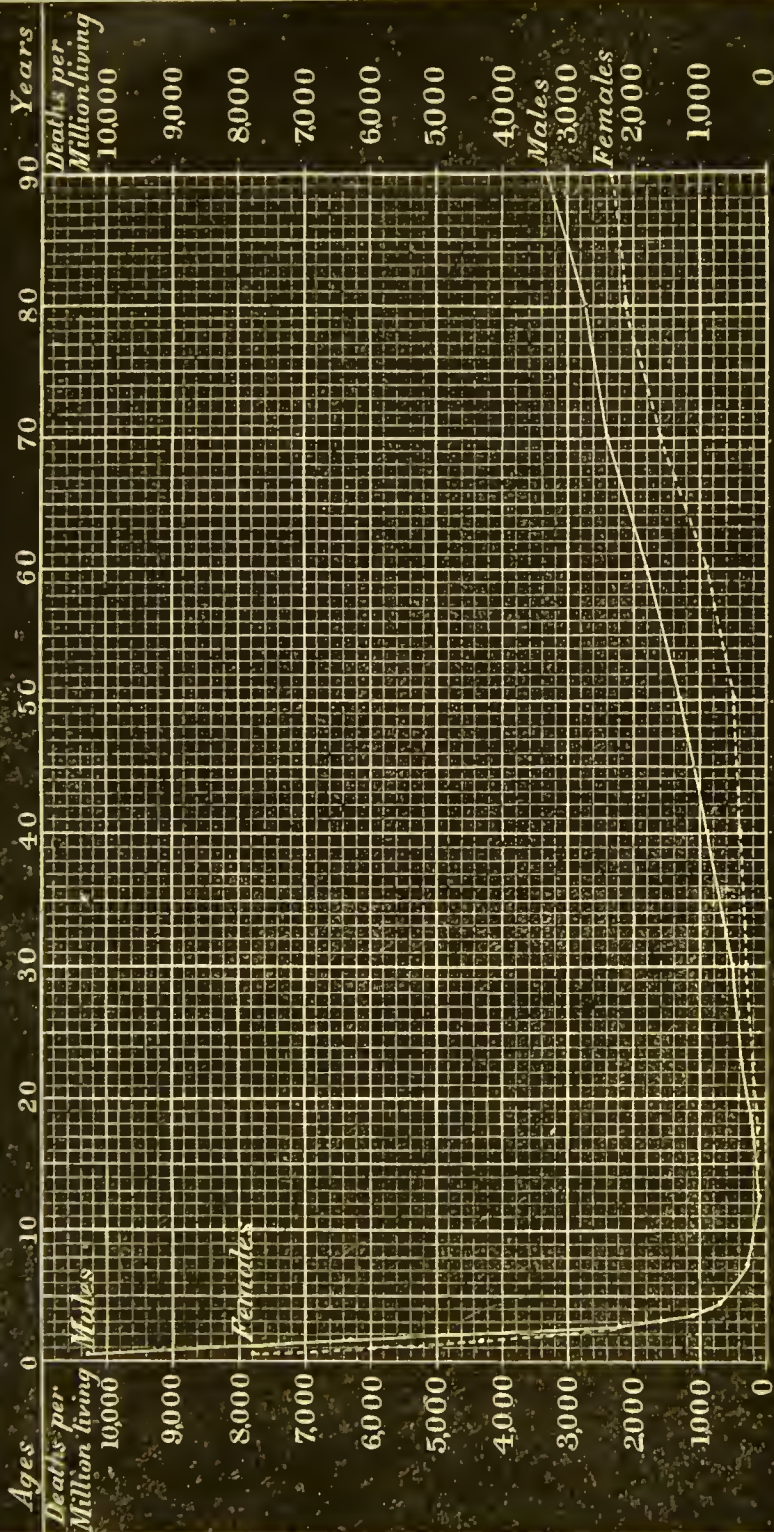
MEMORANDUM ON THE INCIDENCE

## DEATHS from PNEUMONIA in ENGLAND and WALES.

Males.																			Females.																		
	All Ages.	Under 1	1	2	3	4	Total under 5	5	10	15	20	25	35	45	55	65	75	85																			
1871	-	12,776	4,213	2,016	756	337	213	7,535	377	126	189	233	673	741	951	891	733	286	41																		
1872	-	11,031	3,699	1,861	672	315	196	6,743	286	85	151	255	639	865	907	761	642	262	35																		
1873	-	13,233	4,811	1,914	694	349	190	7,328	303	119	197	288	750	1,016	1,097	1,004	792	301	38																		
1874	-	14,990	3,873	2,062	841	362	248	7,366	424	152	266	385	933	1,255	1,448	1,397	978	325	50																		
1875	-	15,968	4,032	2,052	731	366	221	7,406	392	153	271	473	1,101	1,464	1,565	1,544	1,127	407	65																		
1876	-	14,085	3,779	1,925	712	344	239	6,990	349	140	238	359	970	1,194	1,249	1,302	966	349	39																		
1877	-	13,672	3,535	2,050	710	360	219	6,874	336	131	199	340	881	1,210	1,298	1,206	858	289	53																		
1878	-	14,131	3,848	2,106	759	429	219	7,301	400	127	227	310	801	1,127	1,216	1,220	917	364	61																		
1879	-	15,721	3,780	2,182	822	409	252	7,445	460	160	272	391	960	1,418	1,530	1,473	1,092	458	53																		
1880	-	14,666	3,225	2,055	707	417	217	6,621	464	134	278	424	948	1,391	1,450	1,444	1,020	391	41																		
Total -	-	140,822	38,165	20,227	7,404	3,688	2,205	71,689	3,791	1,327	2,288	3,458	8,665	11,681	12,678	12,242	9,095	3,432	476																		
Average -	-	14,082	3,817	2,023	740	369	221	7,169	379	133	229	346	867	1,168	1,268	1,224	910	343	48																		
Rate per 1,000,000 living at each age -	-	1,188	10,589	6,335	2,248	1,149	698	4,353	259	101	195	335	508	881	1,271	1,796	2,442	2,726	3,292																		
1871	-	9,992	3,221	1,914	742	369	232	6,478	370	130	143	168	389	417	432	566	581	280	38																		
1872	-	8,651	2,743	1,675	646	347	184	5,595	317	97	133	167	369	378	372	483	481	226	33																		
1873	-	9,671	2,959	1,782	647	305	166	5,799	317	138	105	189	465	492	508	603	591	280	44																		
1874	-	10,928	2,965	1,816	791	434	240	6,252	383	169	181	229	578	643	612	758	733	340	50																		
1875	-	11,193	2,849	1,815	714	374	208	5,960	391	143	208	284	730	699	727	800	812	380	59																		
1876	-	10,497	2,794	1,777	729	350	238	5,888	344	161	221	217	532	660	565	710	749	315	54																		
1877	-	10,024	2,664	1,830	704	350	193	5,741	369	143	178	210	514	608	537	642	705	322	55																		
1878	-	10,632	2,786	1,920	609	384	253	6,152	378	143	165	237	516	589	545	723	738	387	59																		
1879	-	11,453	2,634	1,919	752	424	258	5,687	441	144	198	239	645	821	720	895	917	379	77																		
1880	-	10,560	2,497	1,859	731	392	255	5,644	442	152	191	234	579	694	598	794	780	393	59																		
Total -	-	103,511	28,022	18,307	7,265	3,729	2,253	59,576	3,752	1,420	1,783	2,174	5,317	6,001	5,616	6,964	7,080	3,302	528																		
Average -	-	10,351	2,802	1,831	726	373	225	5,958	375	142	178	217	532	600	562	696	708	330	53																		
Rate per 1,000,000 living at each age -	-	828	7,801	5,724	2,199	1,155	710	3,613	256	109	150	191	286	415	517	921	1,623	2,074	2,270																		
Mean Population -	-	11,849,418	360,816	319,325	329,142	321,021	316,758	1,647,661	1,459,699	1,311,570	1,176,491	1,032,136	1,705,568	1,324,405	997,999	681,568	372,570	125,841	14,581																		
Males -	-	12,493,034	359,186	319,878	339,144	322,985	316,818	1,649,010	1,467,262	1,300,785	1,187,331	1,134,358	1,861,449	1,431,548	1,087,587	755,902	436,248	159,108	23,347																		
Females -	-																																				



*Average Death Rates from Pneumonia in England & Wales  
Decade 1871-1880.*





## ON THE MICRO-ORGANISMS OF PNEUMONIA.

BY GEO. M. GILES, M.B., F.R.C.S., SAN. CERT. LOND.

IF we open a copy of the College of Physicians' nomenclature of diseases, we find heading the list, as Class I., Sub-division A., a series of general diseases, including the eruptive and paroxysmal fevers, together with cholera, influenza, erysipelas, &c., those known to-day, in fact, as zymotic diseases. It has always appeared to me that this is the only "natural order" in the classification, the remainder of the groups being thrown together on the artificial ground of the localities they may affect.

It would doubtless be difficult to suggest a better method of classification, nor is it the object of this paper to discuss or criticise the College nomenclature. I would merely draw attention to the strong family likeness that runs through the members of the group.

Were one to draw up a "diagnosis" for the class, as is done for plants and animals, it might run somewhat thus:—Diseases gravely affecting the system generally, and characterized by a peculiarly definite course of symptoms, and the production of local lesions, each characteristic of particular individuals of the class.

It is this wonderful uniformity of course, that makes these diseases form such a natural group. In no other is prognosis so definite and easy.

Who of us can tell whether a catarrh will last two or ten days? But take any member of this class, and we can tell to a day what symptoms will present themselves when it arrives. One may feel almost as sure that a peculiar rash will appear on the fourth day of an attack of measles as that a cruciferous plant will have tetradynamous stamens.

Now, it has long appeared to me that acute croupous pneumonia is a malady which should have been included in this class.

In pneumonia, the sequence of symptoms is as regular, and the course of the pyrexia as definite as in small pox or typhoid fever. The local lesion, equivalent to the rash of the one, or the inflammation of Peyer's follicles of the other, appears here as an inflammation of the lungs.

It is true that this one symptom is so prominent as to have caused, hitherto, all attention to be centred on it, but it is to be noted that, like all members of the acute specific diseases, the pyrexia precedes by several hours the advent of the localized symptoms, and does not follow it as is the case with traumatic inflammations, under which heading, broadly, pneumonia would come, if taken as but a result of cold.

As a military surgeon one sees cases, as a rule, earlier than one's *confrères* in civil life, and it has on several occasions occurred to me to admit a man for no other symptom than high pyrexia, in whom, only after twelve hours or more, the characteristic lung symptoms made their appearance.

Now, on first admission there was, I am positive, no change in the respiratory sounds of these men, not even the precrepitant stage of harsh breathing described by Stokes, nor any greater acceleration of respiration than is usual in fever from other causes. The origin therefore of the pyrexia must be sought elsewhere than in the lungs, and it appears difficult to account for it otherwise than by the assumption that there is a poison of some sort circulating in the blood.

The close similarity between the phenomena of acute croupous pneumonia and those of the specific fevers, has, I am well aware, been noticed by many.

Indeed, I well remember the resemblance being pointed out by my old friend and teacher Dr. Handfield Jones, but I do not remember seeing the point formally advanced.

Now, of late years, the evidence in favour of the zymotic origin of the specific fevers, has been gaining strength by great strides.

Starting with the discovery of the bacillus anthracis in the splenic fever of cattle, disease after disease has been added to

the list, till at the present date it may, I think, be said that micro-organisms have been observed in every disease of the class alluded to. Scarcely a week passes now, without a notice in the journals of some new discovery or piece of confirmatory observation in this direction.

That many men of great eminence remain sceptical, more especially as to the causal connection between germ and disease, is but what is to be expected, and yet few surely would be willing to assert that the cause is an inadequate one.

When we see the appalling poisons that can be brewed from a few drops of blood or meat infusion, by the agency of septic bacteria, it is difficult to doubt that poisons of the same kind may be produced in the blood, when some member of the same genus of plants is found growing and active in the circulation of living animals.

That the very existence of these bodies as parasites should be doubted by some, is not so surprising as might, at first sight, appear, for most of them are of such extreme minuteness that their detection, even under high powers, is often by no means easy. Take *e.g.*, the bacillus malariae—a considerable interval elapsed between its discovery in Italy, and the appearance of any confirmatory observations; and, were it not for the worthlessness of negative evidence generally, one might have almost taken it as proved that Prof. Tomassi Crudelis had been in some way misled. Now, however, the malarial-microphyte may be considered an established fact, and its presence has been noted by scores of observers in all parts of the world where paludal fevers are common. The accumulated negative observations are accounted for by the fact that the bacillus is present during only the early part of the paroxysm, and even when present is rather easier to overlook than otherwise.

I have sometimes seen it particularly well, under some chance peculiarity of illumination, and then an accidental movement of the mirror has rendered it perfectly invisible, and, perhaps, half an hour of manœuvring the mirror, would be required before one could again obtain a satisfactory view of it. In fact, the exhibition of these organisms is much more a question of illumination than of magnifying power.

On this account little importance is to be attached to obser-



vations of a negative character in the field of research; and I believe that to the above "diagnosis" might be added the further character—that all these diseases are of parasitic origin, and are caused by poisons elaborated by microscopic plant organisms, which in some way have found a congenial home in the blood and tissues of their hosts.

It was a consideration of the many points of resemblance between the specific fevers and pneumonia that first led me to attempt to ascertain if they agreed in this point also.

In February, 1882, I was stationed at Dera Ismail Khan. There was at the time something very like an epidemic of pneumonia amongst the native troops stationed there, a large number of cases having occurred in each of the three corps that formed the garrison. At the time, however, of my joining the 4th Punjab Infantry, there remained in the hospital only one case in the acute stage.

On examining his blood under a power of about 800 diameters, there were found in it numbers of spherical particles less granular and more refracted than the faint outlined irregular particles so commonly found when great tissue changes are in progress. They were unlike anything I had before met with in blood; but, at the time, I was inclined to think them of extraneous origin, and it was only afterwards that their constancy in several cases, after taking all possible precautions as to cleanliness, led me to impute to them quite another significance.

With the view of testing the question of the possibility of the disease being communicated by infection, I performed one or two experiments, a description of which will be found in the Appendix to the A. M. D. report for 1881, pp. 317, *et seq.*

As far as those observations go, they appear in favour of the possibility of such an occurrence.

The point I would wish more especially to notice here, is that in the blood of rabbits so infected were to be found the very same bodies I had noticed in the human subject.

Since that date two other cold seasons have passed, and a large number of cases have come under my observation, and in all, whether natives or Europeans, these bodies have been present in greater or smaller numbers throughout the course of the case.



The micrococci make their appearance very early, and may be seen—at any rate in small numbers—coincidentally with the earliest symptoms.

When seen in the earliest stage they are all of one form. In cooled blood they appear as minute spherules about 1-27,000th inch in diameter; all are at first free, and appear to contain no nucleus.

If examined on a warm stage, however, the outline appears less round. It is difficult to satisfy oneself as to the true outline of such minute bodies, but they gave me the impression of being pear-shaped. When the examination is made sufficiently quickly, they will be seen to be in free motion, vibrating rapidly to and fro (Brunonian movement), and at the same time shifting their places in a way that can only be accounted for on the assumption that they have the power of automatic movement. More than once, just as the blood was beginning to coagulate, I have “glimpsed” faint indications of a lash springing from the smaller end. This would make the entire body much like a spermatozoon.

My warm stage was, however, a very rough affair knocked up on the spot, and only occasionally acted in a satisfactory manner, so that I have not been able to satisfy myself thoroughly as to the conditions of these bodies in warm blood.

Many appeared to show only the Brunonian vibration, and in a day or two from the outset of a case, numbers of them must necessarily have lost the power of automatic motion, for, by then, many may be seen united in chains of from two to ten individuals.

In this state they are very nearly spherical, or perhaps a very little elongated.

After a day or two, a second larger form makes its appearance. This second form is always nearly spherical, and contains a distinct nucleus.

Coincidentally, or a little after the appearance of these larger nucleated cells, a third form appears, consisting of what appears to be congeries of the smaller kind, united together by a hyaline material. These masses reach the size of 1-1,000th inch or more, and are, I take it, the zooglœa stage of the micrococcus. The material which unites them has a certain amount of firm-

ness, certainly greater than that of the red blood corpuscles, for when these run against the masses, the former alone alter their form. I have very little doubt but that all three forms are but different stages of one and the same organism. In fact, the nucleated kind vary in size from that of the small form to three or four times their diameter, and one can often trace the change that takes place.

The small form appears to be but a nucleus without any visible surrounding protoplasm. When this first becomes apparent it forms a ring round the nucleus, so narrow that it may easily be missed unless carefully sought for: indeed, many individuals that one puts down at first sight as belonging to the first form, on more careful focussing, prove to be nucleated. The bodies united in the zooglœa masses are very like the smaller form. Both free forms stain readily with most anilin dyes, but I have not been able to effect this satisfactorily for the zooglœa stage. The dyes that proved most serviceable in my own hands were anilin blue and gentian violet. The stained specimens retain their colour very fairly after treatment with acid nit. fort part i. water, parts iii.

About the fifth day of the attack there is usually a diminution in the number of organisms floating free in the blood, the absence of the smaller kind being especially noticeable.

This change is, however, only temporary, for the next day they will be probably met with in numbers perhaps greater than ever.

I have thought that, in some cases, I could trace the occurrence of a repetition of the above cycle of changes. Further experience has, however, led me to doubt this. At any rate, after the first four days, the process becomes confused, more or less of all three forms being, as a rule, constantly present.

There is a further peculiarity of these bodies that has, I believe, much to do with the causation of the symptoms they produce, and this is, that they exhibit a strong tendency to adhere to the red corpuscles.

In the earlier stages this tendency is not so marked; but as the case proceeds it becomes more so, and is especially noticeable in serious cases when at their most critical period. Under such circumstances the corpuscles often look like mulberries, from

the number of micrococci sticking to them. Moreover, the latter may be seen, when knocked off by one corpuscle running against another, to quickly attach themselves to the next with which they may come in contact; and I am inclined to ascribe the blocking of the capillaries, which is often to be met with in rapidly fatal cases, to the matting together of the corpuscles thus rendered rough and irregular.

It is certainly remarkable the difficulty with which a specimen of blood is squeezed from the skin, when a patient is in this state; a difficulty decidedly greater than is met with in cyanosis from other causes, and due, as I believe, to the difficulty with which the corpuscles glide over each other, and their so becoming interlocked in the smallest capillaries, which it will be observed, accounts also for the selection of the lungs as the most prominent point of congestion, for there the capillaries are much finer than elsewhere, and the entanglement of the corpuscles most likely to occur.

Amongst natives, cases not unfrequently terminate fatally with a rapidity that vies with that of cholera. One of my own cases died within twelve hours of the first onset. Now these cases died certainly not for want of efficient lung surface to expose to the air, but from obstruction to the circulation, so that the blood could not pass to be aërated. Here the obstruction cannot be caused by cyanosis, as the advent of the former is prior to that of the latter.

The micrococci are formed in varying numbers in the blood, from the earliest stages until the patient is far on towards convalescence. After the acuter symptoms however are passed, their tendency to adhere to the red corpuscles is by no means so marked as at first, and in cases of recovery, at the end, they are only met with free in the blood.

On *post mortem* examination they are to be met with in all parts of the body. The greatest number, however, are to be met with in the lungs.

A section of hepatized lung, when appropriately stained, appears literally made up of little else. The clot within the blood-vessels, the secretion filling the alveoli, and the tissues of the larger septa, simply swarm with them. In pleuritic effusion, if any be effused, they may be found in great numbers, and may be studied here to great advantage.



In sections of the solid organs they are met with in two situations; (*a*) as a constituent of blood clot in the interior of the vessels, and (*b*) in the intercellular lymph spaces. Thus situated, they were found in the liver, spleen, brain, kidneys, skin, and in fact in every organ examined.

They may be cultivated with great facility on slices of boiled potato, after the method described by Koch.

My apparatus consisted of a small basin of porous earthenware, covered with a piece of glass and standing in a plate containing a little water. Occasionally accidental foci of other micro-organisms would appear, but there always sprang up round the point of sputum or blood, a growth of creamy consistence and greyish white colour, the individual elements of which agreed morphologically with the smaller and larger forms above described. The cultivated organism proved capable of infecting rabbits, but was apparently less virulent than the fresh sputa, for recovery followed in each case. The attack thus produced gave, however, no immunity from the action of the original poison.

In the Punjab, especially on the frontier, pneumonia, always sporadically present, not unfrequently presents all the characteristics of an epidemic. There is moreover a very general impression amongst medical officers who have served long there, that the disease is occasionally communicated from one man to another; and although bearing only indirectly on the subject of micro-organisms, I feel sure that the following extracts from a letter on the subject will prove of interest to the readers of the Record.

The writer, Surgeon-Major C. P. Costello, of the 5th Punjab Cavalry, says:—

“Just previously to this (March 1875) the two regiments affected had been marching through the lower Dera Khat and salt districts, at a time when an epidemic of pleuro-pneumonia was raging amongst the cattle there. The 1st Punjab Infantry marched to be stationed in Dera Ghazi Khan, the 5th Punjab Infantry to Ablottabad. In the 1st Punjab Infantry (the corps under Dr. Costello’s immediate observation) out of a strength of 550 there were between 30 and 40 deaths in a very short period; while in the 5th Punjab Infantry the mortality was about a third higher. In the 1st Punjab Infantry, the cases



occurred almost exclusively in two companies, and in the married quarters, the remainder of the regiment keeping almost free. . . . All the signs of a very infectious disease were present ; it spread from one man in each bungalow to another ; from one member of a family to another ; and to the hospital attendants, including one hospital assistant."

It will be admitted that this is a strong piece of evidence in favour of the view that pneumonia may occasionally be communicated by infection.

That it is always so no one would advance, but, however transmitted, I have little doubt that the causation of the disease is to be found in the presence of the above described micro-organisms.

That under certain circumstances they should acquire an increased virulency is quite characteristic of the whole class of zymotic diseases ; and, indeed, in the case of the ordinary putrefactive poisons the process has been imitated experimentally.

A change, in fact, occurs in the characteristics of the ferment, tending to the production of a new species. When we reflect on the rapid sequence of generations that takes place in plants so lowly organised as these, it becomes less surprising that one should see take place, in a few weeks, changes analogous to those which in higher structures require geological ages for their consummation. The change, perhaps, hardly amounts to the evolution of a variety, and would doubtless tend under previous conditions to revert to its previous state ; but a change there is, as evidenced by the increased activity of the disease produced, and one which no doubt better fits it to live under the conditions in which it finds itself.

There indeed appears something remarkably illustrative of the versatility of these organisms, in the circumstances under which they may be cultivated by "Koch's method."

To breed a vegetation, accustomed to develop in the blood of a living mammal, on scraps of potato, on gelatine, and what not, seems, at first sight, much like attempting fish culture out of water. The experiment, however, is in many instances notoriously successful. The consideration naturally follows, that if they develop so well under two such utterly different surround-

ings, may there not be many others which may be equally congenial? the soil, for example. Further, is it not highly probable that the parasitic condition, under which it has forced itself to our notice, may be, after all, quite foreign to its usual habits?

In earth or water, immense numbers of organisms of this class may be met with, some of them practically indistinguishable from those above described, and, without in any way asserting their identity, the habits of these bodies make it a quite possible contingency. Taking this supposition as true, pneumonia would depend on this plant finding in certain individuals a suitable soil in an unexpected situation.

That only certain individuals should be found offering a *pabulum* on which the plant can thrive, is only what is observed in all zymotic diseases, and depends, no doubt, on the system of those attacked being in an abnormal condition. It remains only to consider in what this abnormality may consist. In the case of pneumonia, I believe this will be found to be the state brought about by repeatedly breathing the same air, and that the only reason why pneumonia is more common in winter than summer is because in the former season folk are apt to abjure ventilation.

Viewed in this light, the discrepancies, noticed in the B. M. J. retrospect for 1883, between Friedlander's experiments and those made by Sulvioli Zäslem, and myself, are seen to be of little import. My dogs were recently captured pariahs, who had lived a free open-air life; his had probably been tied up for some time in some out room of his laboratory. Accordingly his inoculations in canine subjects were successful, and mine failed. On the other hand, my rabbits, though to all external appearance healthy, had been long breathing an atmosphere which any one who has ever smelt a rabbit hutch must recognise as hardly sanitary; while the rabbits which he failed to infect may have been recently captured, or for some other reason in good sanitary condition.

Morphologically, the distinctions between the different ferment organisms is small, and one is strongly drawn to the conclusion that many of the recently discovered disease germs may be but varieties of common soil and water organisms. Doubtless, when a markedly infectious disease has been evolved, the

variety has become a more or less permanent one, and would then probably rebel against being grown on so foreign a soil as a slice of potato offers.

At least I do not remember seeing anywhere any notice of such an experiment proving successful with the germs of small-pox or measles.

Given that bad ventilation may produce in the blood and excretions a soil suitable for the development of the pneumonia ferment, it is likely enough that the produce of the cells bred in this new situation would be better fitted for thriving there than the original parasites; and it is quite conceivable that in the course of generations a fitness might be reached which would make it vie with the plague in infectiousness. That pneumonia rarely becomes infective is probably due to the circumstance that most people allow themselves a spell of ventilation in summer, whereby the supply of suitable *pabulum* is cut off from the plant, or that the raw parasite, unaccustomed to its work, has to start anew on its course towards infectiveness during the next winter.

Such an idea may appear far fetched and fanciful to many, but there will, I think, be this much found in support of it, *viz.*: that it is to be noticed that the most of the instances that have been published of transmission of pneumonia by infection have occurred at the end of the winter.

At any rate, the difference between the organisms of disease and those found everywhere in earth and water are so small that it must be conceded that they cannot have diverged far from some common parent-stem.

While making some check observations in connection with these pneumonia organisms, I met with, in cases of mumps, a micrococcus very like that of pneumonia. A want of further cases has prevented my following up this chance observation, but I saw sufficient of them to feel sure that it would have been difficult to distinguish the one from the other in two unlabelled slides.

In cases, again, of ordinary malarial fever, at the end of the hot stage, may be observed particles (the spore stage, I take it, of the now well known bacillus) which are scarcely distinguishable from the small form of the pneumonia ferment.

The course of further development here leads to marked differences, but the critical stage appears nearly identical.

There is another remarkable point in connection with Dr. Costello's instance of epidemic pneumonia—the coincidence of epidemic pleuro-pneumonia amongst the cattle of the district. Without explicitly stating it, he evidently suspects that there was some connection between the outbreak amongst the animals and that in man.

It would be interesting to know if any other instances of the same kind have been observed, especially when taken in connection with Dr. Kröbner's observation as to the similarity of the microphytes found in the two diseases.

In conclusion, I may say that the deductions I would draw from the above considerations are :

1. That pneumonia is a zymotic disease.
2. Under certain conditions the disease appears as an epidemic.
3. When epidemic it may become infectious.

I am aware that some of these deductions rest on but scanty evidence, and must apologise for perhaps pursuing imaginary consequences too far. I shall be satisfied, however, if I can induce others to follow up the subject, and confirm or correct the views I have taken.

That I may have to modify these, I regard as more than probable; but, whatever may be the fluctuations of individual ideas and evidence, I feel sure that the great broad facts on which rests the zymotic theory of disease will but develop to greater and greater certainty.



## FIRST REPORT ON PUERPERAL PYREXIA.

### METHOD OF CLASSIFICATION.

THE number of cases included in the present first Report on puerperal pyrexia is 354. For the analysis of these cases, it has been thought desirable to classify them on a uniform principle, namely, according to their causation, so far as it has proved possible to infer this, with a reasonable degree of probability, from the Returns. In doing this, it has been found necessary to leave a large class, Class VI., containing 113 cases, in which the causation could not be definitely assigned from the account received. In a considerable number of other cases, it appeared doubtful to which, out of two or more of the classes, they most properly belonged. Cases of this kind have been placed at the end of the class which on the whole appeared the most appropriate, as doubtfully belonging to that class.

The following is the scheme according to which the cases have been classified.

### PUERPERAL PYREXIA TABLES.

- I.—Local origin.
- II.—After difficult labour.
- III.—Originating in or after exposure to contagion.
  - a.—From zymotic diseases
    - 1. Scarlatina.
    - 2. Erysipelas.
    - 3. Enteric.
    - 4. Measles.
    - 5. Rötheln.
    - 6. Variola.
  - β.—From other cases of puerperal septicæmia.
  - γ.—From septic material.
    - 1. Post-mortem poison or discharge from wounds.
    - 2. Insanitary conditions.
  - δ.—Fever commencing before delivery, but no infection known.
- IV.—Cold or exposure.
- V.—Shock or emotion.
- VI.—Cause not assigned.

In cases where causation by contagion is suspected, it is of course impossible that there can ever be absolute proof that any individual case actually originated from the contagion. Hence in the sections corresponding to the several zymotic diseases of Class III., Group *a*, first of all those cases have been placed which actually showed the symptoms of the zymotic disease; next, those in which it appeared clear from the history that there had been exposure to the contagion; and finally, those in which even the exposure appeared to be somewhat uncertain, or to have occurred at a considerable interval before delivery. Other cases, again, in which the possibility of contagion appeared still more remote, have not been placed in the zymotic class at all.

Taking the main division of puerperal pyrexia into autogenetic and heterogenetic cases, it will appear that the autogenetic group corresponds in general to Classes I., II., IV., and V. in the scheme, the heterogenetic group to Class III. It is, however, of course possible that in some cases ranked under Class II. as originating after difficult labour, there may have been contagion in addition. Again, in Classes IV. and V., the exposure or the emotion, which appeared to be the immediate antecedent of the symptoms, may in some cases have supervened upon some other cause of puerperal pyrexia.

In Class I., that of cases of pyrexia due to local origin, those cases alone have been placed in which there was a definite history of some local condition sufficient to account for the pyrexia, such as adherent placenta, or retention of decomposing clots which were afterwards expelled. The occurrence of an offensive lochial discharge by itself has not been considered sufficient ground for placing a case in this class, since it is probable that decomposition of the lochial discharge may sometimes be only secondary to contagion. It is probable that a large number of cases really of local origin are included in Class VI., that in which the causation could not be definitely assigned. Class II., that of cases occurring after difficult labour, does not include all cases in which forceps were applied, since some of these can probably hardly be regarded as cases of difficult labour, and some of them appeared to be more appropriately placed under different headings. Section 2, in Class III., Group *γ*, including

pyrexia occurring after exposure to insanitary conditions, is a large one, comprising 49 cases. Insanitary conditions existed in other cases besides these, but there appeared to be more positive reasons for classifying these elsewhere. The presence of insanitary conditions should probably be regarded as, in many cases, a predisposing cause, rather than as proved to be the actual exciting cause of the pyrexia.

#### RELATIVE MORTALITY.

A comparison of the mortality in some of the various classes may afford some indication as to whether they correspond to any real or essential differences in the forms of pyrexia. The average mortality in the whole number of cases is 47·4 per cent. If Class III., comprising the probably heterogenetic cases, be excluded, the mortality in the remainder, or probably antogenetic cases, is only 37·7 per cent., while in Class III. it is as great as 59·4 per cent. In Class II., that of cases occurring after difficult labour, it is 50 per cent. Taking the sections of Class III. separately, and excluding cases doubtfully classed, we have the following results. In cases of actual scarlatina, mortality 25 per cent.; in cases occurring after exposure to contagion of scarlatina, but not showing the symptoms of that disease, mortality 57·1 per cent. In cases of actual erysipelas (including cases 189, 215), mortality 37·5 per cent.; in cases occurring after exposure to contagion of erysipelas, but not showing the rash of that disease, mortality 83·3 per cent., the highest mortality recorded in any class or section. In cases occurring after exposure to contagion of enteric fever (3 cases only), mortality 66·6 per cent. In cases of measles, r  theln, and variola (2 cases only), no mortality. In cases ascribed to the contagion of other cases of puerperal pyrexia, mortality 65 per cent. Thus while the recorded cases of actual scarlatina and erysipelas, as well as those of measles, r  theln, and variola, have not a high mortality, those which occurred after exposure to the contagion of scarlatina, erysipelas, enteric fever, and puerperal septic  mia all show a mortality very much greater than the average. A separate group, namely, Class III., Group  $\delta$ , has been formed to include those cases in which no source of infection was known, but in which the fever commenced before delivery, since it has been

thought that this circumstance probably indicates that the disease was due to some septic or zymotic poison. In this section also the mortality is a very high one, namely, 5 in 6, or 83·3 per cent., a rate equal to that in the cases which occurred after exposure to the contagion of erysipelas.

CASES OF LOCAL ORIGIN OR OCCURRING AFTER DIFFICULT  
LABOUR. CLASSES I. AND II.

In Class I. (that of local origin), the mortality is 33·3 per cent. or rather less than the average mortality of all cases probably autogenetic. It is hardly necessary to seek for evidence to show that local causes, such as decomposition of retained placenta or clots, may originate puerperal pyrexia. But it is in this class, if anywhere, that we may expect to find some evidence as to the results of local treatment, or bearing upon the question whether, in the presence of an offensive lochial discharge, not only vaginal but uterine injections are desirable. There are several cases in which marked benefit is recorded as having followed quickly upon the use of uterine injections, or upon the removal or expulsion in some way of the offending cause. In case 5, on the 10th day there were severe rigors, and the temperature was 105° F. The uterus was then examined, and a small piece of firmly attached placenta peeled off. Recovery was now rapid. In case 7, on the 3rd day, temperature was 105° F. Stinking clots were expelled, after gentle compression of the uterus, and vaginal injections. Temperature fell the same evening, and recovery was rapid. In case 9, in which lochia were very offensive from the first, uterine injections were used first on the 4th day, and daily after that. The symptoms gradually abated after the 4th day, although the temperature became normal only on the 17th. In case 11, on the 19th and 20th days, clots, and a putrid tumour the size of a pullet's egg were passed. On the 21st day, temperature and pulse were normal. In case 16, there was a rigor on the 5th day, T. 103·8° F., P. 125, lochia suppressed. A clot was then removed from uterus by finger in cervix with hypogastric pressure. Lochia returned freely in 10 hours; and all symptoms now steadily improved. Case 42 was first seen on the 7th day. The uterus was then syringed out every 4 hours. On the 8th day, tempe-



perature had fallen from 104° F. to 102° F., and from this time there was steady improvement.

Vaginal injections seem to have been used in most of the cases in this class, uterine injections are mentioned in 12 cases (2, 8, 9, 14, 18, 22, 28, 32, 34, 38, 41, 42). The mortality in these is 4 or 33·3 per cent., precisely the same as the average of the whole class. This result seems to be in favour of intra-uterine injections, since they would probably be used in the worst cases. We have ourselves met with a case showing the value of uterine injections perhaps more strikingly than most of those here recorded. A primipara had severe rigors little more than 12 hours after delivery. When seen at the end of 24 hours, lochia were suppressed, P. 160, T. 105° F., the patient delirious. The case had all the aspect of the most virulent form of puerperal pyrexia derived from contagion. Since, however, careful inquiry failed to indicate any possible source of contagion, we decided on washing out the uterus, though there was no evidence of decomposition. The fluid used was a solution of iodine (Tr. iodi ʒij. ad aq. oj). After 12 hours, temperature had fallen to normal, pulse to 90, the lochia had returned, and the patient from that time did well.

The cases in Class II., occurring after difficult labour, may be regarded as analogous to those in Class I., since local inflammation is the probable origin of the pyrexia. It is possible, however, that in some, the local lesions may simply have facilitated the reception of some heterogenetic poison. In case 52, a possibility of contagion from other puerperal cases is indicated. In several other cases (47, 51, 59) there is a record of a rash, which may raise the suspicion that there may have been some zymotic poison concerned.

#### CASES ASCRIBED TO ZYMOTIC OR SEPTIC POISONS. CLASS III.

##### ERYSIPELAS.

Cases of erysipelas, or ascribed to the contagion of erysipelas, will first be analysed, since erysipelas is the zymotic disease, the connection of which with puerperal pyrexia is most generally admitted.

There are eight cases in which erysipelatous rashes occurred, namely, cases 98 to 103 and cases 189, 215. In three only of these,

namely, cases 100, 101, 102, there is a history of probable exposure to the contagion of erysipelas. The mortality in the 8 cases is 3 or 37·5 per cent. In two cases (100 and 101) the rash extended from the vulva, in three cases (98, 99, 103) it affected the thighs or legs, having begun in case 98 from a varicose ulcer two days before delivery. In case 102, where erysipelas had been in the house 14 days previously, and the patient had washed the linen of an erysipelatous patient, the rash was a scarlet efflorescence general over body except face and chest, slightly raised, patchy round knees, varying much daily in colour, vividness and extent, not followed by desquamation. In case 189 the rash was upon face, neck and arms; in case 215 on the face, secondary to glossitis. The latter case is of interest in reference to a possible relation between the poisons of different zymotic diseases. The patient had a sore throat with plastic exudation (?diphtheritic) 48 hours before delivery. Five hours after labour acute glossitis set in, and seven hours later facial erysipelas commenced. On the sixth day there was phlegmasia dolens of left leg with erythema, spreading to foot, This was followed by abscesses on the dorsum of the foot and in the calf, and on the 19th day there was still greyish plastic lymph in the throat. The case ended in recovery.

With regard to the question whether the poison of erysipelas in these cases affected specially the peritoneum or pelvic organs, we have the following information. Of the two cases (100 and 101), in which the rash extended from the vulva, in case 100, there was distension of abdomen, pelvic inflammation, uterus enlarged and tender; in 101, the abdomen was distended, the uterus enlarged, and the result was fatal, though the erysipelatous rash amounted only to a slight flush about the wound of a ruptured perinæum. In case 98, commencing two days before labour, the uterus and lochia were normal, but the right shoulder became inflamed, and the result was fatal. In case 215 also there was no local pelvic inflammation, but in all the other cases there were some local symptoms. Thus in case 99, there was vaginitis and abdominal distension. In case 102, the lochia were scanty, the abdomen tender and tympanitic. In case 103, there was tenderness of uterus, scanty offensive lochia, probably pelvic inflammation, and in case 189, metritis

at first and slight pelvic inflammation, lochia profuse and foetid. Thus foetor of the lochia is noticed in two of the eight cases.

Two of the eight cases appeared to be the source of infection. Case 101 appeared to be the origin of case 111, in which the symptoms were those of puerperal peritonitis, without evidence of erysipelas. In case 99, the child died from erysipelas shortly after death of mother. This case is of interest from the fact that the symptoms of pyrexia commenced after 48 hours, but the large hard red painful swellings in arms and legs appeared only on the seventh day. No origin of contagion of erysipelas was traced, but the accoucheur was attending several cases of enteric fever. An adherent placenta was removed by hand.

In considering the cases referred to the contagion of erysipelas (104 to 115), the most striking point is the very high mortality, namely, 10 in 12, or 83·3 per cent. This would seem consistent with the view that the poison of erysipelas may directly affect the peritonæum, and be then more dangerous than erysipelas of any external part. Case 113 is the only one in which the contagion of erysipelas was conveyed to others. In this, the child had erysipelas of hand and wrist one week later. From two cases there was probable or possible conveyance of infection to other puerperal women. Case 107 gave rise to a slight epidemic, but no other case was fatal. After case 114, the attendant had another fatal case of puerperal peritonitis at an interval of five days.

In all the cases in this group there was abdominal distension or other evidence of peritoneal or pelvic inflammation.

#### SCARLATINA.

Thirteen cases (61 to 73) appear to be clearly cases of scarlatina from the evidence of the rash; there was also sore throat, or redness of fauces, in all of these cases except one, namely, case 61. The mortality is not very high in proportion to the general average of puerperal pyrexia, namely, 4 in 13, or 30·7 per cent. With reference to the possibility of contagious origin of cases of puerperal pyrexia in which no contagion can be traced, it is of interest to note that in three of these cases (67, 68, and 73) there is nothing recorded to account for



the contagion, and in five others (61, 62, 66, 69, and 72), it is only noted that scarlatina existed in the neighbourhood at the time. Scarlatina was conveyed to others in one case only—case 63. In this the child had scarlatina; also six children in the next house, of whom two died. In case 69, there is a curious history. On the seventh day after patient's delivery, the husband, who had been constantly in sick room, developed diphtheria with dense membrane and albuminuria, no rash. On the ninth day, sister developed diphtheria and slight erythematous eruption on chest. On the twenty-first day, mother, who had nursed all three, had erysipelas, which began on face, and later became general. Insanitary conditions existed in this case.

It is still a matter on which opinions differ whether the poison of scarlatina can produce a disease resembling ordinary puerperal peritonitis, and unaccompanied by characteristic rash or sore throat. In reference to this question there may be some significance in the fact that local inflammation of pelvis or peritonæum is recorded in most of these cases of actual scarlatina. In one case only, case 70, uterus and lochia were normal; in case 72, the uterus was normal, but lochia slightly offensive, calling for uterine injections. In all the rest there was some local inflammation, and in all the fatal cases the abdomen became distended. In one case, case 64, left inguinal parametritis followed. Of the thirteen cases of scarlatina, it is noted in four cases (61, 62, 63, 67), that the lochial discharge was scanty, and in five (61, 63, 65, 66, 72), that it was offensive. There is therefore some evidence from these cases that an offensive lochial discharge, which is generally considered to be in most cases primary, and the cause of puerperal pyrexia, may, in some instances, be secondary to contagion.

Besides the thirteen cases which have been classed as being pretty evidently cases of scarlatina, there are seven others which might also be considered to be most probably cases of that disease, but in which the rash was not so characteristic, namely, cases 17, 29, 47, 267, 289, 295 and 308. Of these, there was sore throat in four cases only (17, 29, 267, 295). In one of these only (289), a possible source of infection is mentioned, in the fact that the accoucheur was attending cases of scarlatina



at the same time. In case 267 both the nurse and husband had sore throat. Of these six cases, in two there appeared to be a sufficient local cause to account for the pyrexia. In case 17 a large decomposing clot was expelled from the uterus. In case 29, the pyrexia was ascribed to too frequent examinations by the midwife, and the lochia were foul. Again, in case 47, there was a lacerated perinæum. The probability that the disease was really scarlatina varies in the different cases, and the view apparently taken by the reporter has generally been followed in the classification. The question, whether the disease may not have been scarlatina arises also more or less in all the cases referred to in the preceding section.

The question has been discussed whether, in cases of puerperal septicæmia, a rash may occur, more or less resembling that of scarlatina, and leading to an erroneous assumption of the presence of that disease, or, on the other hand, whether a rash not obviously that of scarlatina or erysipelas may indicate that the disease is really of zymotic origin. It may therefore be of interest to review those cases in which rashes are recorded. Setting aside the cases ranked as scarlatina, there was a general scarlet rash in eight cases, including six out of the seven just referred to (17, 29, 47, 59, 214, 267, 289, 295). Of these there was sore throat also in five (17, 29, 214, 267, 295). The mortality in the eight cases was four, or 50 per cent. In one of them (214) pneumonia had commenced three days before delivery, and was apparently the cause of death. There was a roseolous, papular, or petechial rash in ten cases (4, 8, 77, 110, 121, 175, 192, 271, 308, 316), not including cases of merely sudamina or miliary eruption. Of these cases 4 and 8 have been regarded as of local origin, occurring after removal of adherent placenta. Case 77 is attributed to contagion of scarlatina; case 110 to that of erysipelas; case 121 occurred after exposure to contagion of enteric fever. In cases 175, 192, there were insanitary conditions in the house. In the remainder the cause of the pyrexia could not be assigned. Sore throat was combined with the rash in only two cases (77, 175), and in both it was slight. The mortality in the ten cases is very high, namely, 8 or 80 per cent.

An erythematous or scarlet rash occurring in limited blotches is noted in six cases (51, 52, 147, 159, 259, 322). Of these cases

51 and 52 occurred after difficult labour; case 147 was possibly due to the contagion of puerperal septicæmia; case 159 to that of pyrexia; cases 259 and 322 are placed in the uncertain class (VI.), but possible contagion of cellulitis is suggested in 259, of diphtheria in 322. The mortality in the six cases is again very high, namely five, or 83·3 per cent. So far then as the evidence of these cases goes, it would seem that those forms of rash, if they do not indicate a zymotic origin of the disease, at any rate imply a severe and dangerous form of puerperal pyrexia.

In the group of cases ascribed to the contagion of scarlatina, but not showing definitely the signs of that disease, 24 cases are placed, 11 of them doubtfully. In some of these there were symptoms more or less suggestive of scarlatina. There was sore throat without rash in five cases (75, 76, 78, 82, 83). In case 77, there was raised papular rash on chest and abdomen at end of first week, no desquamation, slight sore throat some days after rash. In case 78, besides sore throat, there was desquamation during convalescence. In case 79, though there was no sore throat at first, there was later sloughing of fauces and tonsils. In all these cases there was marked pelvic or peritonitic infection. Positive scarlatina conveyed to others is noted in one case only, one in which there was neither sore throat nor rash, namely, case 90. In this, the child was at breast till fifth day, and had scarlatina two days after mother's death. Besides this case, the child died on the sixth day in case 84; the child died jaundiced on second day in case 97; a nurse had bad sore throat in case 95. The mortality in the group is very high, namely 64 per cent.; excluding cases doubtfully classed, it is 57·1 per cent.

Taking erysipelas and scarlatina together, it may be said that the view now accepted by most, that contagion derived from erysipelas or scarlatina occupies an important place in the causation of puerperal pyrexia not manifestly showing the signs of the specific disease, receives strong confirmation from two facts in the records now collected. The first is the considerable number of such cases which are attributed by the reporters to the contagion of these diseases. The second is that among the cases so attributed the mortality is so much higher than the

average of puerperal pyrexia. It seems impossible to explain this circumstance, if it be a false assumption to assume that any link of causation exists. The fact that, in several cases of manifest erysipelas and scarlatina, local lesions in pelvis and peritonæum were noted, is also a point in favour of the view that in other cases due to the same poison such local lesions may be the chief or only manifestations. The relative mortality in the different groups appears to point to the conclusion that a woman who is infected by the poison of the zymotic disease is in less danger when the ordinary signs of the disease are manifested than when they are not.

#### ENTERIC FEVER.

In accordance with the general opinion that erysipelas and scarlatina are the zymotic diseases chiefly to be considered in reference to the causation of puerperal pyrexia, very few cases are referred to the contagion of other specific diseases. Three cases, two of them fatal, occurred after exposure to the contagion of enteric fever (cases 121, 122 and 123). From one of these (123) there was a possible conveyance of contagion, as the attendant had three other cases of puerperal peritonitis, two of them fatal, within the following month.

#### MEASLES.

There are two cases of measles after delivery, ending in recovery (cases 124, 125). In case 124 the uterus and lochia were normal; in case 125 the uterus was very tender and lochia foul, but here there was a small pelvis, instrumental labour, child stillborn, and lacerated perinæum. There is one case (126) of puerperal pyrexia having the usual symptoms of puerperal peritonitis, occurring after exposure to the infection of measles. It ended in recovery after three months. It was the first of a series of five similar cases, all ending in recovery.

#### RÖTHELN.

Case 127 is recorded as one of severe rötheln after delivery. The uterus and lochia were normal, but there were acute abscesses of ears. Case 129 seems to be possibly also one of rötheln, and, in case 128, local pelvic inflammation occurred



after exposure to the contagion of rötheln. All three cases ended in recovery.

## VARIOLA.

There are two cases of variola, both ending in recovery (130, 131). In both of these local pelvic symptoms are noted. In case 130 pelvic inflammation was followed by general peritonitis, and, at the beginning of the second week, cellulitis in the left broad ligament. There is a history of possible conveyance of the contagion by the accoucheur. The disease commenced on the third day, and the variolous eruption was preceded by general scarlatiniform efflorescence. In case 131, premature delivery at seven months took place two days after the appearance of the variolous eruption. The uterus was large and somewhat tender. The child died a few days later with obscure symptoms of blood poisoning.

Besides the cases which have been classed as more or less probably due to the contagion of zymotic diseases, there are others in which such contagion is mentioned as a possibility. Thus the contagion of scarlatina is mentioned as a possibility in the following cases,—75, 125, 133, 160, 175, 179, 193, 209, 212, 234, 247, 253, 276, 302 and 308; that of erysipelas in the following—136, 160, 197, 202, 243 and 258; that of variola in 185. In case 177, in which the symptoms were those usual in puerperal peritonitis, and which is classed as due to insanitary conditions in the house, the child died from erysipelas four weeks later.

## DIPHTHERIA.

There is no section of cases occurring after exposure to the infection of diphtheria, since there are none in which the history appeared to be sufficiently definite to justify such a classification. A possibility of such contagion is, however, suggested in cases 179, 211, 267 and 322. In only one of these (267) did a slight sore throat occur. Both the nurse and husband also suffered from sore throat. In case 322, on the 17th day a crimson erythematous rash appeared on the abdomen. There were also purpuric spots on trunk and extremities, and death occurred on the 20th day. The patient three months



before delivery had nursed two children with fatal diphtheria and purpuric eruption. Of the four cases (179, 211, 267 and 322) three were fatal.

It is an interesting and unsettled question whether the plastic exudation in the throat or in the vagina, which is sometimes seen in cases which are apparently cases of ordinary puerperal peritonitis, especially in epidemics in lying-in hospitals, is to be regarded as evidence of true diphtheria, and, if not, whether it has any relation to that disease. On this point little information is to be gained from the cases included in this Report. A diphtheritic or pseudo-diphtheritic exudation in the throat is noted in only five cases (176, 188, 215, 223 and 283). These do not seem to have been cases of very exceptional severity, for recovery took place in three out of the five, or 60 per cent. In cases 176 and 215, there was no local pelvic inflammation. In the rest there was abdominal distension, or signs of pelvic inflammation. Cases 176 and 188 are attributed to insanitary conditions in the house. In case 215 there was sore throat (? diphtheritic) 48 hours before delivery. This was followed by glossitis, erysipelas, and local abscesses, and the case has been already mentioned under the head of erysipelas (see page 123). Case 223 is attributed to the patient's having gone down stairs with insufficient clothing soon after delivery. Case 283 is attributed by the reporter to intemperance during puerperal state. The exudation is described as clear lymph (not diphtheritic) on uvula, soft palate, fauces, and pharynx. No contagion to others from any of these cases is recorded.

In case 53, occurring after difficult delivery with forceps, with slight laceration, there was a sloughy condition of the vagina which had a diphtheritic appearance on the fourth day. Febrile symptoms commenced on the second day, and death occurred on the sixth.

Sore throat is also noted in the following cases, besides those which have been already mentioned in connection with scarlatina—17, 29, 30, 43, 51, 74, 75, 77, 78, 104, 119, 172, 175, 200, 201, 207, 210, 214, 225, 295, 317. In these 21 cases the mortality is 10, or 47·6 per cent., a mortality higher than the general average. Of these, the sore throat was associated with general erythematous or scarlet rash in cases 17, 29, 214; with

erythematous blotches in case 51, with papular rash in cases 77, 175; with desquamation of skin in case 78.

## CONTAGION FROM PUERPERAL SEPTICÆMIA.

In Class III., Group  $\beta$ , that of probable infection from other cases of puerperal septicæmia, 24 cases are placed. The mortality of these is very high, namely, 17, or 70·8 per cent. In 7 of the 24 cases the conveyance of contagion is ascribed to a midwife. There is no record of further contagion conveyed from any of these cases to other puerperal women, but, in case 142, the child died on the twelfth day with abscesses in different parts. An offensive lochial discharge is noted in 8 of the 24 cases (133, 134, 135, 141, 147, 151, 152, 153). Besides the cases placed in this group, the possibility of infection from puerperal septicæmia is suggested in cases 255, 256, 269.

## POST-MORTEM POISON.

Only three cases are recorded (161, 162, 163), in which there was a possibility of contagion from post-mortem poison, and one only of these (163) was fatal. Five cases (156, 157, 158, 159, 160) may have been due to contagion from the discharge of wounds, the two last cases being doubtfully placed in this class. Of these only one (158) was fatal.

## INSANITARY CONDITIONS.

In the section of cases ascribed to insanitary conditions, namely, Class III., Group  $\delta$ , Section 2, 49 cases are included, 8 of them being doubtfully placed in the section. The mortality is comparatively low, 34·1 per cent., but if the doubtful cases, in some of which there was suspicion of contagion from other sources, be included, it is raised to 42·8 per cent. Only those cases have been placed in this class in which insanitary conditions were in the house itself, not merely in the neighbourhood. Attention may be called to case 201, in which, when the patient was in a very grave condition, rapid improvement followed her removal, on the sixth day, to another house. Again, in case 174, improvement began on the closure of an untrapped sinkhole near door at foot of stairs. In case 189, after abscesses of both breasts with sloughing of skin and cellular tissue, erysipelas of

the left side of head and face occurred at a late stage. In case 177, a fatal case, there was contagion of erysipelas to the child. This is the only case in the section in which contagion to others is reported. Diphtheritic throat was observed in two cases in the section, 176 and 188.

No special sections have been formed for cases probably due to the poison of gonorrhœa, but this origin, in conjunction with exposure to cold, is suggested in case 230. In this case empycæma occurred, and death on the 18th day.

#### FEVER COMMENCING BEFORE DELIVERY.

Group  $\delta$ , in Class III., that of cases in which fever commenced before delivery, but no source of contagion is recorded, is a small but interesting one. It comprises six cases, with a mortality of 5 or 83·3 per cent., equalled only by the mortality of cases ascribed to the infection of erysipelas, but not showing any erysipelatous rash. In two of the cases (214, 217) the disease was pneumonia, commencing in case 214 three days, in case 217 one day before delivery. Considering the resemblance which pneumonia has to a zymotic disease, it is of interest to note how far the pelvic organs were affected in these cases, and whether they have any points of likeness to ordinary puerperal septicæmia. In case 214 there was inflammation of the uterus and in the pelvis, and the abdomen became distended, but the lung affection appeared to be the direct cause of death. Case 217 is one of great interest. There was no sign of local inflammation in uterus or pelvis, and no distension of abdomen, although occasional diarrhœa occurred. The reporter, however, considers that the fever of the pneumonia really developed into a condition resembling ordinary puerperal pyrexia for the following reason:—On the fifth day after delivery he was called directly from visiting this case to another confinement. This second case was speedily over, and the patient went on well till the fourth day, when she had a rigor, and afterwards developed all the symptoms of puerperal pyrexia, and finally died five days later. He paid also a single visit, without making examination, two days after delivery, to one of many cases attended by assistant. This patient had disagreeable symptoms, and temperature up to  $102^{\circ}$  for several days, while all the assistants



other cases did well. The reporter gave up midwifery practice for six weeks, and had no more bad cases afterwards. In case 213, the patient had nursed husband with severe tonsillitis. Symptoms of peritonitis with vomiting came on near the full term of pregnancy, labour took place three days later, and death occurred five days after delivery. In case 216, the reporter considered that the fever, which commenced three days before delivery, might possibly be enteric. Case 215 may be one of primary diphtheria before delivery, succeeded by erysipelas after delivery, and eventually pyæmic abscesses. This is the only case in the group which ended in recovery. The nature of the febrile attack in case 218 is obscure. A temperature of  $105^{\circ}$  and pulse of 150 were coincident with an abortion at the third month. Next day pulse and temperature were normal, but on the 4th day there was a rigor with return of pyrexia, ending in death on the 15th day.

The following are cases in which there was evidence of pyæmic abscesses or inflammation:—8, 26, 77, 127, 128, 142, 162, 183, 193, 195, 215, 230, 271, 348, 352. In the following there was secondary pneumonia:—2, 26, 59, 78, 90, 129, 132, 193, 194, 203, 214, 216, 217, 221, 247, 250, 320, 322, 331, 351.

#### COLD OR EXPOSURE.

In thirteen cases there appears to be a definite history of origin after cold or exposure. Four other cases are doubtfully placed in this class. The mortality is 23·5 per cent., or 30·7 per cent. if doubtful cases be excluded. In case 223, there was a diphtheritic sore throat; in case 225, slight congestion of throat. No pneumonia or bronchitis is noted in any of the cases, except case 226, where there had been a bronchial catarrh for one week previous.

#### SHOCK OR EMOTION.

It is well known that mental shock or emotion may cause a temporary rise of temperature and pulse in a puerperal woman. If the six cases included in Class V. are rightly ascribed to the influence of such a cause, it appears that not only temporary pyrexia but pelvic inflammation and even death may result, for two of the six cases proved fatal. It may be open to



question, however, whether there may not have been also in such cases some local or other cause kindled into greater activity by the mental impression. It will be observed that the onset of the disease was generally later than usual, being on the fifth or some later day in four out of the six cases.

#### CONTAGION TO OTHERS.

There are 19 out of the 356 cases from which a probable or possible conveyance of contagion to others is recorded, but this was to other puerperal women in eight only out of the 19. The eight cases are cases 3, 99, 105, 114, 123, 126, 217 and 315. Of these, case 3 is ascribed to local origin, the next five are in different sections of Class III., which comprises all cases probably heterogenetic. In the last two cases the causation is not determined. The other cases of possible communication of contagion are the following:—In case 51, the nurse had sore throat for two days. In case 63 the child took the contagion from puerperal scarlatina. In case 84 (attributed to contagion of scarlatina) the child died on sixth day. In case 90 (one in the same section) the child had scarlatina two days after mother's death. In case 95 (also in the same section) the child died one week later of pyæmia; the doctor's hand was severely poisoned; of two nurses, one had had sore throat, the other a sharp attack of facial erysipelas. In case 97 (also in the same section) the child died jaundiced on second day. In case 99 puerperal erysipelas was communicated to child. In case 142 (ascribed to contagion from puerperal septicæmia) the child died with abscesses in different parts. In case 177 (ascribed to insanitary conditions) the child died with erysipelas in four weeks. In case 267 nurse and husband had sore throat. In case 332 child died from pyæmia. Thus, in nine cases, the child appears to have received contagion. About all the 19 cases, which were the possible source of contagion, the most striking point is that all were fatal except case 126. Thus, so far as the evidence of these cases goes, there is support for the view commonly held that it is chiefly from fatal or very severe cases of puerperal pyrexia that contagion is to be feared.

The opinion has been held by some that no contagion is possible from autogenetic cases, but only from those originating

in some zymotic poison. On this point the present records do not afford decisive proof. Case 3, indeed, appears to be one of local origin from complete laceration of perinæum associated with offensive lochia. But case 151, to which contagion may have been conveyed, was attended a month later, and the reporter had attended 8 cases in interval without bad result. The evidence of any connection is therefore uncertain. This point is one of those on which evidence may be hoped for from future collective investigation.

## POINTS FOR FURTHER INQUIRY.

Amongst other points calling for further inquiry may be mentioned the question whether the poison, not only of erysipelas or scarlatina, but also of enteric fever, diphtheria, variola, rötheln, measles, or pneumonia, may give rise to a disease resembling puerperal septicæmia, the possibility of which occurrence is suggested by a few cases now recorded. Further information is also to be desired as to the mode of recognizing those cases from which contagion is to be feared, or chiefly to be feared. With regard to treatment, it has not been found possible to draw any certain conclusions from the record now published.

---

The Committee is indebted to Dr. GALABIN for the preparation of this Report, and to Mr. O. A. BROWNE for editing the Returns which follow.

## APPENDIX OF RETURNS ON FEVER IN THE PUERPERAL STATE.

The enquiry issued upon Puerporal Pyrexia ran as follows:—

### No. VII.

#### FEVER IN THE PUERPERAL STATE.

*Will you kindly answer the following questions about the last, or any other, case of Pyrexia after labour of which you have an accurate record?*

Observer's Name.....  
Address.....  
Date at which Case occurred.....

- 1.—What circumstances may have predisposed to the disease?
- 2.—Can you trace it to infection from any specific fever, septic influence or other cause?
- 3.—Was there a rash? If so, where?  
What were its characters?  
Was there sore throat? If so, what were its characters?  
" " distension of abdomen?  
" " local inflammation in pelvis?  
" " " " elsewhere?
- 4.—What was the condition of the uterus?
- 5.—How soon after labour did the fever begin?
- 6.—What was its duration?
- 7.—What was the result?
- 8.—Give a brief history of the course of the disease and the treatment.

A shortened abstract of the 354 cases reported upon is hereto appended in the form of the replies received to each of the questions numbered as above.

Negative replies have been omitted, where not considered of importance to the case.

The cases have been classified according to their probable cause, upon the following scheme:—

- I.—Local origin.
- II.—After difficult labour.
- III.—Originating in or after exposure to contagion.
  - a.—From zymotic diseases
    1. Scarletina.
    2. Erysipelas.
    3. Enteric.
    4. Measles.
    5. Rõtheln.
    6. Variola.
  - β.—From other cases of puerperal septicæmia.
  - γ.—From septic material.
    1. Post-mortem poison, or discharges from wounds.
    2. Insanitary conditions.
  - δ.—Fever commencing before delivery, but no infection known.
- IV.—Cold or exposure.
- V.—Shock or emotion.
- VI.—Cause not assigned.

This classification is not to be considered as in all cases rigidly correct. Many of the cases are assigned only to such cause as has appeared upon the whole to be the most probable.

For the sake of clearness, some of the Abbreviations used are given below, together with the word they represent.

y.=year; m.=month; w.=week; d.=day; h.=hour; D.=death; T.=temperature; P.=pulse; R.=respiration; Sl.=slight, slightly; prev.=previous, previously; infl.=inflamed, inflammation; dist.=distension, distended; suppd.=suppressed; del.=delivery; Ut.=uterus; Vag.=vagina, vaginal; injn.=injection; tym p.=tympanites, tympanitic; p.p.=post partum; nat.=natural.

## CLASS 1.

## LOCAL ORIGIN.

NAME AND ADDRESS OF OBSERVER.	NO. OF RETURN (for identifica- tion only.)
Elsom, F. J., L.R.C.P., Chesterfield, Derby . . . . .	1
Fell, Walter, M.B., St. Thomas's Hospital . . . . .	2
Hooker, Chas. P., L.R.C.P., Cottishall, Norfolk . . . . .	3
Berry, Wm., L.R.C.P., Wigan . . . . .	4
Fisher, Fred. B., L.R.C.P., Dorchester . . . . .	5
Fiddian, Alex. P., M.B., Cardiff . . . . .	6
Eddowes, Alfred, M.R.C.S., Market Drayton . . . . .	7
Mitchell, T. Alex., L.R.C.P., Catford, S.E. . . . .	8
Blake, G. F., M.R.C.S., Birmingham . . . . .	9
Bowen, O., M.R.C.S., Liverpool . . . . .	10
Rae, Geo. A., L.R.C.P., Devonport . . . . .	11
McLean, Allan, M.B. Ed., Portland, Dorset . . . . .	12
Sheen, Alfred, M.D., Cardiff . . . . .	13
Barfoot, G. H., M.D., Birkenhead . . . . .	14
Workman, Chas. J., M.D., Teignmouth, Devon . . . . .	15
Pilcher, W. J., F.R.C.S., Boston, Lincoln . . . . .	16
Waugh, John, M.D., Toddington, Beds . . . . .	17
Buckell, Arthur E., M.D., Chichester . . . . .	18
Armstrong, H. G., M.R.C.S., Reading . . . . .	19
Lynch, J. Roche, L.R.C.P., 8, Boyne Terrace, Notting Hill, W. . . . .	20
Brown, John, L.R.C.P., Bacup, Lancashire . . . . .	21
McNaught, Jas. W., M.D., Nurchurch-in-Rossendale . . . . .	22
Dalton, Henry W., M.D., Dublin . . . . .	23
Wilks, George, M.B., Ashford, Kent . . . . .	24
Startin, James, M.R.C.S., 16, Sackville Street, W. . . . .	25
McMurray, W., M.D., Templepatrick, co. Antrim . . . . .	26
Colbeck, Thos. W., L.R.C.P., Dover . . . . .	27
Johnson, C. J. B., L.R.C.P., Wetherby, Yorks . . . . .	28
Kershaw, Alfred, M.R.C.S., Farnworth, Bolton . . . . .	29
Franklin, Geo. C., F.R.C.S., Leicester . . . . .	30
Bernard, Walter, M.R.C.S., Londonderry . . . . .	31
Leary, Thomas, L.K.Q.C.P., Castlederg, co. Tyrone . . . . .	32
Thompson, E. T., L.K.Q.C.P., Wolston, Coventry . . . . .	33
Brady, Chas. M., L.R.C.S., Wigan . . . . .	34
Bain, William, L.R.C.P., Heaton Chapel, near Manchester . . . . .	35
Mahomed, Geo., M.R.C.S., Bournemouth . . . . .	36
Coombs, C. P., M.D., Castle Cary, Somerset . . . . .	37
Hedley, John, M.R.C.S., Middlesborough . . . . .	38
Finzi, J. M., L.R.C.P., 99, Sutherland Gardens, W. . . . .	39
Hawkins, Cæsar F., M.R.C.S., Clifton . . . . .	40
Crawford, James, L.K.Q.C.P., Ightham, Kent . . . . .	41
Alderton, H. C., L.R.C.P., Barnoldswick, Leeds . . . . .	42

- 1.—1. Former bad labours. 2. Placenta prævia. Ch. dead in utero.  
 3. Abd. distd. 4. Ut. flabby. 5. 3rd d. 6. 6th d. 7. R.  
 8. Occas. hæmorrhage 1 mo. before labour. Placenta removed by hand,  
 coming away in parts; all removed at time of labour. Injns. used and  
 opium given internally. 3rd d. T. 102, P. 120, feeble; abdom. tenderness,  
 tympanites, vomiting; no sleep. 4th d. T. 103, P. 140. Opium, and salines  
 given. 5th d. T. 104, P. 140. Improvement from 6th d.



2.—2. Placenta intimately and universally adherent. Excessive p. p. hæmorrhage. 3. Abd. distd. Pneumonia. 4. Ut. large, not v. tender. 5. 3rd d. 6. 9 d. 7. D. 8. 3rd d. rigor. T. 105. tongue dry, loch. offensive. 4th d. cough with rusty sputum, and scattered crepitation over left lung at back; never distinct dulness or tubular breathing. T. about 104 throughout; delirium last 3 d.; no diarrh. or vomiting.

Gave ferri perchlor., large doses quinine, Warburgh's tincture; daily ut. injns.; stimulants; good diet.

Attending many patients, but no similar case, either before or since.

3.—1. Unmarried. House small and dirty. 3. Abd. much distd. Pelvic inflammn. 4. Ut. hot and tender. Lochia offensive. 5. 3rd d. 6. 4 d. 7. D. 8. Aged 17, attended by midwife; perinaeum lacerated; large opening from vag. into rectum. Veetis used later. 3rd day, peritonitis set in. D. on 7th d.

Treatment, for peritonitis. Injns. used.

(Infection possibly conveyed to case 151.)

4.—1. Unmarried. Mental depression. 3. Roseola over sternum. Sl. tympan. distn. of abd.; gen. abdom. tenderness. 4. Ut. — 5. 60 h. 6. 84 h. 7. D. 8. Primip. lab. nat. Placenta sl. adherent, removed by 2 fingers in os, with abdom. compression. Next day clot expelled. 3rd d. rigor, tongue dry and coated, tenderness over whole abd. Opium, poultices; sponging. 5th d. Pain less; loch. ceased; skin hot, tongue dry and brown. 6th d. P. 130, thready; abd. tympanitic and v. painful. 7th d. got up. On getting back into bed fell back and died suddenly.

5.—1. Sl. constant pain over uterus during pregnancy. 3. Abd. not distd. 4. Ut. contained small piece of adherent placenta and membrane. 5. 2nd d. 6. 10 d. 7. R. 8. 2nd d, patient felt ill, T. rising. 10th d. sev. rigors. T. 105. Ut. examined and piece of placenta found firmly attached; this peeled off; small, very adherent piece of membrane left. Recovery now rapid.

Treatment, quinine; vag. injns. Later, iron.

6.—1. Insan. conditions. 2. Labour at 4th month. Adherent irremovable placenta. 3. Abd. distd. Gen. peritonitis. 4. Ut. enlarged, with few small ragged threads marking site of placenta; tissues at this point black and thin. 5. 1. w. 6. 1. w. 7. D. 8. Under chloroform attempt made to remove placenta, but without success. 8th d. small portion of black flocculent membrane passed after syringing; sharp rigor with severe vomiting and pain, at same time.

Treatment, opium, benzoate of soda; vag. injns. P. M. Some pints of pus found in abdomen. Intestines matted together. Ut. empty; no pus in cavity. Placental site marked by fibrinous shreds implanted over orifice of L. Fallopian tube.

7.—2. Decomposition of contents of imperfectly contracted ut. 3. Sl. distn. of abd. 4. Ut. large and tender. 5. at once. 6. 4-5 d. 7. R. 8. Primip; long exhausting labour; forceps used. 3rd. d. T. 105, ut. large and tender. Stinking clots expelled after gentle compression of ut. and vag. injections. T. fell same evening. Catheter used many days. Recovery rapid.

8.—1. Jaundice, 7 y., 3. y., 2 y., previously. 2. Placenta adherent at upper and back part, removed by hand introduced into ut. 3. Rather prominent bright red isolated papules over abd. Abd. distd.; pelvic inflamm. Sl. pericarditis. 4. Ut. swollen, hard and v. tender. 5. 48 h. 6. 4 d. 7. D. 8. 2nd. d. loch. suddenly arrested. T. 104, Resp. hurried; pulse quick and irregular; breath offensive. 3rd. d. delirium with suicidal tendency. 4th d. coma and death. T. never above 110°.

Treatment, quinine, opium, and calomel; turp. stupes, linseed and mustard poultices to abd.; ut. injns. Brandy freely.

- 9.—1. Anæmic. 2. Laceration of cervix and perinæum. P. p. hæmorrhage. 3. Abd. not distd. 4. Cervix lacerated; involution slow. 5. 72 h. 6. 16 d. 7. R. 8. Ushered in by sl. rigor and fever, quick pulse and respiration, intense frontal headache, anorexia, insomnia. For several days lay semiconscious, but no delirium. Lochia v. offens. from first. Milk scanty. Gradual abatement of symps. after 4th. d. T. normal on 17th d.

Treatment, vag. injns. twice daily from the first; ut. injn. on 4th d. and daily after that. Quinine, digitalis, opium, iron, arsenic, phosphorus, nux vomica, given in succession; brandy, champagne, and nutritious diet.

- 10.—2. Primip. tedious labour. Att. by midwife, who had to tear away the placenta, as it was perfectly adherent. Rather profuse p.p. hæmorrhage. Husband had pneumonia in same bed 1 mo. previously. 3. Abd. sl. distd. with gen. tenderness. 4. Ut. tender. 5. Next day. 6. 11th day. 7. R. 8. 2nd d. T. 103, P. quick, great thirst and anorexia; gen. abdom. tenderness, increased by slight hacking cough, which continued for next 8 d. No signs in chest at any time; no expectoration. Small doses of quinine and tinct. opii given. Improved daily till 9th d., when T. 105, P. 130 compressible; vomiting everything she took. Vomg. at once stopped by efferv. ammonia draught. 10th d. T. 101·4, had slept. Now progressed favourably. T. normal on 11th day.

- 11.—2. Tumour left in uterus. Had attended enteric case (1st wk.) and case of measles. 3. Miliaria on R. cheek. Herpes on L. ala nasi. Abd. distd. 4. Ut. enlarged for few d., not tender. 5. 10th d. 6. 11 d. 7. R. 8. Primip. 24; forceps used; p.p. hæmorrhage. Did well till 9th d. when sat out 2 h. Same evening T. 102. 12th d. even. T. 103·6. Large doses of salicylate given. 13th d. T. 103·1 with profuse persps. Quinine in small doses given. For next 4 d. T. 100 to 101·5. 19th d. large clot passed. T. normal. 20th d. further clots passed; also putrid tumour the size of a pullet's egg. 21st d. T. and P. normal.

- 12.—2. Placenta retained 40 h; removed with difficulty; consid. p.p. hæmorrh. 3. Abd. distd. Gen. peritonitis. 4. Ut. normal. Loch. offens. for 1st few days. 5. 4th d. 6. 4 d. 7. D. 8. Primip. 19, attended by midwife. Placenta firmly adherent; manually removed under chloroform piece by piece. 3rd d. Large piece of placenta came away. 4th d. Rigors, high fever, delirium; gen. abdom. pain and swelling; milk suppd.; symps. of collapse.

Treatment, diaphoretics, opium; hot fomentns; vag. injns. Many confinements in previous and following week, but no bad symps. (This the only case in 1119 confinements during last 10 y.)

- 13.—2. Partial placenta prævia, with much hæmorrhage. Version for hand presentation. 3. Abd. not distd. 4. Ut. large and painful. Lochia never offensive. 5. 2nd. d. 6. 6 d. 7. D. 8. 2nd d. sev. rigor. 3rd d. M. T. 105, P. 168. Ev. T. 104, P. 130, tongue dry; ut. large and painful; no local peritonitis; lochia suppd.; no milk. Small doses aconite, poultices, vag. iujns. ordered. 4th d. T. 106·2, P. 172, vomiting; B.O thrice. In evening, T. 100, P. 130, perspiring, feeling better. 5th d. Rigor. T. 102·4, P. 160. perspiring. Quinine given. 6th d. T. 100·8; much vomiting. 7th d. Bad night, rigors. T. 103·2, vomiting, jactitation, delirium. Ev. T. 99·8. 9th d. Delirium, constant vomiting of blackish matter. Death.

Attended 3 other cases in next 3 w.; no further child-bed fever.

- 14.—2. Insan. conditions. Premature labour at 7th m.; adherent placenta. 3. Sl. sore throat, aphthæ. Abd. distd. Some pelvic cellulitis. Phlebitis both femoral veins. 4. Ut. imperfectly contracted 5 w. 5. 36 h. 6. 3 w. 7. R. 8. Labour natl. and easy. Placenta generally adherent, removed by hand. In 36 h. T. 105, lochia fetid. Uterino injns. given with pills of opium quinine and digitalis. Phlebitis of both femoral veins occurred within 3 w. Sl. purulent discharge from ut. for next 6 w. Recovery very slow. One y. later, miscarr. at 3rd m., placenta again retained.

15.—2. Attended by midwife. Membranes retained. Sore throat epidemic (? catarrhal diphtheria). 3. Abd. distd. Pain in L. iliac fossa. 4. Ut. large. 5. 3rd d. 6. 10 d. 7. R. 8. 3rd d. fever; abd. distd. and v. painful; loeh. foul. Some membranes removed, and vag. injns. given. Treatment, benzoate of soda, Dover's powder, then opium and bark. Recovery rapid.

16.—2. Sitting up on 5th d. Chill. Lochia suppressed. Rapidly changing clot removed from ut. 4 h. later. 3. Sl. distn. of abd. Great tenderness over lower  $\frac{1}{2}$  of abdomen. 4. Ut. distd., tender. 5. 5th d. 6. 4 d. 7. R. 8. Lab. easy. Chill on 5th d. 5 h. later T. 103·8. P. 125. R. 20.; great tenderness over lower abd.; lochia suppressed. Clot removed from ut. by finger in cervix with hypogastric pressure. Perceptible relief followed removal. Subsequent treatment, opium; turp. stupes; rectal and vag. injns. Lochia returned freely in 10 h. and all symps. now steadily improved.

17.—2. Retained clot. 3. Diffuse erythematous rash on chest and becoming general; no visible desquamation. Gen. redness of pharynx; no membrane. Abd. distd. and tympanitic. 4. Ut. large and tender. 5. 2nd d. 6. 7 days. 7. R. 8. 2nd child. Lab. v. rapid; placenta removed entire. Ut. flabby and slow to contract; ergot given and continued. Vag. injns. used. 3rd d. restless, T. 104·5. P. quick, with rash and sore throat; swelled tongue. Ergot, aconite, pot. chlor. given; vag. injns. continued thrice daily. 4th d. large decomposing clot expelled. 5th and 6th d. muttering delirium during sleep; fever subsiding. T. normal on 7th d. Ragged ulcers on tongue for 1 w. Made good recovery.

18.—1. Poverty and overcrowding. Debility. Consid. post partum hæmorrhage. No med. assistance till 2 h. after del. 2. Retention and foetid decompn. of membranes within ut. 3. Mod. distn. of abd. Chronic brouchitis. 4. Ut. enlarged, tender. Loch. v. offensive. 5. ? Within 48 h. 6. 2 m.s. 7. R. 8. 1st to 10th d. Vomg., severe headache; T. 102 to 104. First seen on 10th d., loeh. v. offensive; extreme pallor; no periuterine infln. felt. Ut. washed out freely. 11th d. mass of decomposed membranes discharged. Next d. T. 105. 16th to 21st d. T. from 104 to 102. 21st to 30th d. T. 103 to 100, with occasional rigors; then gradually fell to normal.

During the fever occas. albuminuria noticed, with œdema of ankles, subsultus tendinum and nocturnal delirium; 2 attacks of purpura. Treatment, ammon. carb; vag. injns; good diet with free brandy. Recovery complete.

19.—2. Lingering labour; forceps used. Placenta adherent, removed by hand. 3. Sl. distn. of abd. Loch. offensive. 5. 2nd d. 6. 4th d. 7. D. 8. 2nd d. rigor, profuse sweating. T. 108; no pain in abd. or pelvis. Died after 4 d. in this condition. No P. M.

20.—3. Consid. distn. of abd. Inflm. affecting R. iliac and femoral veins, with tenderness over ovary of same side. 4. Ut. large and flabby, os patulous; slight black offensive discharge. 5. 2nd or 3rd d. 6. 5 d. 7. R. 8. Had sudden strain 2nd d. after deliv. Large and heavy ut. suddenly anteverted with pain and arrest of loch. for 36 hrs. One hour later, rigor, T. 102·6, P. 120, pain on R. side of ut; abd. prominent; sweating, dull headache, sickness. 3rd d. large decomposed clot passed, followed by black offensive disch. Ut. large and anteverted. Gave small doses quinine and laudanum; fluid diet; port wine. In evg. T. 100. P. 104; sleepless. Gave weak injn. Cond. and water; followed by rigor; patient feeling worse. 4th d. M., T. 101·2. P. 80. Ev., T. 101·2. P. 92. Heavy dull headache; no vomiting or rigors; disch. of darker colour; pain in R. side of pelvis and over iliac vein. 5th d. better night. T. 99·6. P. 88. loch. offensive; less pelvic pain; cramps and numbness in left leg. Veins hard some wks. Quinine continued. No further bad symptom. Good recovery.



- 21.—2. Attended by midwifo. Flooding; probably imperfect contraction of ut. Retention and putrefaction of clots. 3. Abd. not dist. No local infln. 4. Uterus? contracted. 5. 13th d. 6. 40 h. 7. R. 8. Delivery followed by consid. flooding, with vomiting. Able to be out of doors on 12th d. On 14th d. severe vomiting; labour-like pains; offensive clots discharged. Patient extremely pale. P. rapid and weak. T. 103. Frequent small doses, salicin and aconite given. Vag. injns. used. Temp. fell within 48 h. Convalescence gradual. P. continued rapid for 14 d.
- 22.—1. Mental depression. Great dread of this confinement. 2. Placenta adherent, removed by hand introd. into ut. No *direct* infection; no other cases myself, but many fatal in district. 3. Abd. sl. distd.; pelvic infln. 4. Ut. normal. Loch. scanty. 5. 24 h. 6. 11 d. 7. D. 8. In 24 h., fever, sl. pain in pelvis. Local symps mild throughout. Some vomiting early; occas. rigors. T. 105 to 106 in Ev. with marked morning remission. Treatment, quinine in large and small doses, digitalis, tinct. opii; cold applied to head and abdomen; uterine injns.
- 23.—3. Abd. slightly distd. Local infln. in pelvis and in left thigh. 4. Ut. relaxed. 5. 3 w. 6. 12 d. 7. R. 8. Ut. relaxed after del. Loch. free till fever, then ceased. Clots frequently passed; some of them decomposing. Loch. v. foetid 3rd w., when rigors. T. 103. P. 120. Treatment, quinine, salicin.
- 24.—1. Anxiety about confinement. Version in previous delivery. Version performed for shoulder presentation. 3. Abd. distd. 4. Ut. not firmly contracted. 5. 3rd d. 6. 7th d. 7. R. 8. 3rd d., restless, excitable. T. raised. P. quick. 4th d. extreme anxiety and prostration; loch. foul; tympanites, diarrhoea, sweatings; in evening some mental hallucinations. Treatment, antiseptic; sodæ hyposulph., quinine, chloral; turpentine stupes; vag. injns. Good diet with small doses brandy.
- 25.—1. Low state of health before confinement. 2. Accoucheur had attended cases of scarlat. 1 w. previously. 3. Abd. distd. Inflam. in pelvis. Peritonitis. 4. Ut. enlarged, tender. 5. 5th d. 6. 6 w. 7. Puerperal mania; and pelvic abscess. 8. Perinæum lacerated. Legs fastened together to promote adhesion, so parts imperfectly cleansed. 5th d. Rigors, high fever, acute abdom. pain and tension, lochia and milk suppd. P. small and feeble. Anxious expression. Insomnia. Great variations of temp. with muttering often violent delirium followed, till abscess formed in left iliac fossa, pointing in inguinal region. Abscesses opened 3 mos. later. Much foul pus. withdrawn. Recovery slow.
- 26.—1. Hour glass contraction of ut. Retained placenta, removed with difficulty. 2. Abd. distd. Continuous fixed pain in right side of pelvis. Pulmon. congestion. Pyæmia. 4. Hour glass contraction, then slightly enlarged and tender. 5. 2nd d. 6. 19 d. 7. D. 8. 2nd. d. T. 100. continuous fixed pain in R. hypogastrium. Light linseed poultices. Liq. opii sedativ. Ut. washed out. 3rd d. rigor. T. 104. Opium, quinine and whiskey ordered. 4th d. rigors continuing, profuse sweats. Turpentine stupes to abd. Treatment as before. 5th d. Abscess in R. labium incised. Pain in R. ankle. 6th d. pain in shoulder, and on 8th d. in wrist; rigors and sweats continuing. 12th d., joints swollen and v. tender. Opium and quinine continued; sodæ salicyl. at night. 13th d. Aspect good. Joints better. Bases of both lungs congested. Linseed and mustard poultices. Pills stopped. Turpentine given. 18th d. Increased chest symps, dyspnoea, cyanosis. 19th d. diarrhoea. Death.



*(Doubtfully placed in this class).*

27.—2. Probable septic absorption into uterine veins. Attending fatal enteric case at time. 3. Abd. distd. 4. Ut. tender. 5. 4th d. 6. 9 d. 7. R. 8. *Æt.* 46; 7th ch.; labour nat. Catheter 8 d. 4th d. rigor, fever, loch. offensive. 11th d. maxim. T. 103·6. P. 104. 28th d. severe nephralgia; relapse lasting 3 w., during which sl. temporary albuminuria and passage of phosphatic calculus, as also again 2 w. later. Treatment, salines, pot. chlor., quinine, stimulants.; assiduous poppy fomentns., poultices; vag. injns. Pulse long remained rapid.

28.—3. Abd. sl. dist. Uterine infln. Consolidn. of apex of left lung noted on 12th d. 4. Ut. enlarged and sl. painful. 5. 36 hrs. 6. 28 d. 7. R. 8. *Æt.* 35; 3rd ch.; labour easy. 3rd d., noon, T. 100·5. P. 120. sl. pain over uterus. Midnight, T. 103. some pain in heart regn. 4th, 5th, and 6th days T. 104·5 to 100. No cardiac murmur. 7th and 8th days T. 102 to 99·6. T. in left axilla repeatedly 1 deg. higher than in right. 9th to 14th d. T. fr. 103 to 99·4. Slight cough on 9th d. 12th d. apex of left lung consolidated, dulness to 3rd rib. 16th to 28th d. T. from 102 to 98, rising at noon. From 29th d. T. normal; steady convalescence.

Treatment, salines, quinine, salicylic acid, Fellow's syrup; uterine injns.

29.—2. Repeated examns. by midwife. 3. General miliaria, first vesicular, then deeply erythematous, with small imperfect pustules. Soft palate, pharynx, tonsils, congested and purple. Abd. dist. Infln. about uterus and over whole abd. 4. Ut. enlarged and tender. 5. 36 hrs. 6. 11 d. 7. D. 8. Primipara; long forceps used. 2nd d. T. 104 M., 105 E. lochia foul; uterus and adjacent pelvic area painful and tender; abdom. distd., tympanitic and tender. Poultices to abd. Vag. injns. Small doses opium, quinine, and digitalis. 3rd d. T. 102. P. 130. R. 36.

A few days later medicine being rejected, small doses quinine in powder given and retained. Frequent brandy and Brand's extract. T. still 103 to 102. abdom. and pelvic tenderness continuing. 10th d. R. frequent. P. 140. Hypostatic congestion at bases both lungs. D. on 11th d.

30.—1. Anxiety. Dread of confinement. 3. V. slight sore throat. Abdn. dist. Pelvic infln. 4. Ut. normal; os. patulous. 5. 12th d. 6. 10 d. 7. R. 8. Labour easy. 10th d. slight hæmorrh. fr. uterus. 12th d. Loch. scanty, not foul. No local signs. 14th d. P. 108, good vol; some sickness. 15th d. T. 104. P. 140; sickness; abd. distd.; loch. absent. 16th d. some small clots discharged; marked pelvic tenderness. For next 3 d., T. 104 to 101. P. 140 to 120; then gradual improvement.

Treatment, opium, stimulants; vag. injns.

31.—2. Forceps used. 3. Abd. normal. 4. Ut. relaxed and tender. 5. 2nd d. 6. 4 d. 7. R. 8. 3rd d. T. 100. P. 108., ut. relaxed and tender. Injection given. Ergot and Dover's powder to 6 doses. T. normal on 6th d.

32.—3. Abd. sl. tympanitic. 4. Ut. sl. inflamed 8th d. Loch. offensive. 5. 8 d. 6. About 60 d. 7. R. 8. 8th d. severe rigor. T. 103. tenderness over uterus, disappearing in 8 d. Fever for 60 days with rigors every 2nd day; then gradual improvement.

Treatment, large doses quinine, opium, sodæ sulph. carbol; ut. and vag. injns. Nourishing diet; little stimulants.

33.—1. Prev. unhappiness. Delivered by midwife. 2. Drains defective. 3. Sl. distn. of abdn. General abdom. tenderness. 4. Ut. v. tender. Loch. v. offensive. 5. 5 d. 6. 8 d. 7. D. 8. High fever and rapid pulse from commencement. Constpn. at first, then diarrh. Dysuria. No delirium. Died from asthenia on 8th d.

Treatment, opium, quinine, stimulants; vag. injns.

34.—2. 14 fatal puerperal cases in same town, but none under my care. 3. Abd. dist. Cystitis. 4. Ut. congested. Loch. dark and very offensive. 5. 3rd d. 6—. 7 R. 8. Primip. lab. easy. Doing well till 3rd d. Loch. free. 3rd d. Ev. T. 103. abdn. dist., pain and tenderness over ut. Loch. stopped. 4th d. T. 104 M., 105 E. tenderness over bladder, dysuria. 5th d. T. 105 M., 106 E., delirium. 6th d. T. 105 M., 107 E., no change.  $\frac{1}{2}$  gr. doses of antim. tart. given. T. fell to 101, and did not again rise above 102. Hence gradual recovery.

Treatment, quinine (large doses), opiates, sodæ salicyl; poultices; ut. and vag. injns.

35.—1. Anxiety. 2. Leucorrhœa before parturition. 3. Abd. not distd. 4. Ut. normal. 5. 4 d. 6. 2 d. 7. R. 8. 3rd d. T. 101. 4th d. T. 106. Vag. syringed. T. normal in 2 hours, and so remained. Perspns. and weakness several days.

36.—1. Albuminuria after del. 3. Abd. not distd. 4. Ut. painful on 5th d. Loch. odorous. 5. 4 to 5 d. 6. 10 d. 7. R. 8. Lab. rapid. Albuminuria on 5th d. 5th d., T. 103, ut. pain, loch. offensive. Vag. injns. used. 7th d. T. normal. Phlegmasia dolens later.

37.—1. Mental depression; ? Chill. 3. Flatulent distn. of abd. 4. Ut. norm. 5. 24 h. 6. 3 w. 7. R. 8. Rupture of membr. 40 h. before del. 2nd d. catarrhal symps; much uterine pain; opium given. 3rd d. herpes on lips; aconite given. 4th d. Loch. offensive; shred of membr. discharged. Vag. injn. used. 4th to 14th d. T. varying from 98 to 103. Quinine and ergot given. 15th d. Decline of fever.

38.—2. Placenta adherent; removed by hand smeared with lard. 3. Abd. distd. 4. Ut. very tender. Loch. stopped. 5. 2nd d. 6—. 7. R. 8. 2nd d. Rigor. sev. abdom. pain, T. 104. 3rd d. Evg., T. 105.4, restless. 4th d. Ev., T. still high, abd. distd. and pain severe.

Treatment, salicylic acid, opium, quinine; uterine injns.

39.—1. Living over stable. 3. Sl. distn. of abd. Œdema of labia. 4. Ut. painful, tender. 5. 2nd d. 6. 14 d. 7 D. 8. Version for placenta prævia. Began 2nd d. with rigors. and vomiting.

Treatment, aconite, quinine, iron, morphia.

40.—2. Primipara 27, labour short; sl. laceration of perinæum. 3. Sl. dist. of abd. 4. Ut. normal. Loch. offensive. 5. 3 d. 6. 4 d. 7 R. 8. Sl. fever after delivery. 3rd d. T. high, great thirst, perspns. at night. Loch. scanty and offensive. Oppressive sensation at chest.

Treatment, salines, quinine, opium, digitalis; vag. injns.

41.—1. Great anxiety about father's illness. 3. Abd. distd. towards close. Pelvic infn. 4. Ut. soft. 5. 2nd d. 6. 6 d. 7 D. 8. Lab. rapid. Adherent placenta removed by hand introduced into ut; perinæum lacerated. 3rd d. being partially exposed, felt chill and sev. pain in back and above pubes. T. 102, P. 128. 4th d. T. 104. 5th d. T. 104, restless, loch. becoming dark and fetid. 6th d. T. 104 M., 103 E., unconscious. In evening had 2 severe uræmic fits 7th d. T. 102, unconscious, restless; one slight fit. Ur containing  $\frac{1}{2}$  albumen. In evening, T. 104, loch. very dark and fetid. 8th d. Rigor. at noon, T. 105, P. 144. Catheter used. 9th. d. D. exhausted. Abd. much distd.

Treatment, salines, purgatives, bromide of ammonium; ut. and vag. injns. daily.

42.—1. Insanitary conditions. 2. Retained clots. 3. Rash on face, chest, abdomen. (i.) Vesicular. (ii.) Pustular. Sl. distn. of abd. Infiltration on R. side of pelvis. 4. Ut. enlarged, v. tender. 5. 5 d. 6. 14 d. 7 R. 8. Primip. attended by midwife, labour easy. 5th d. sev. rigor. Seen on 7th d. Ut. syringed out every 4 h.; poultices to abd; quinine and opium frequently. 8th d. T. had fallen from 104° to 102°. From this time steady improvement.

## CLASS II.

## AFTER DIFFICULT LABOUR.

NAME AND ADDRESS OF OBSERVER.	NO. OF RETURN.
Ross, Roderick R., L.R.C.P., Stornoway, N.B.	43
Kempe, Arthur, M.R.C.P., Edin., Exeter	44
Garstang, T. W. H., M.R.C.S., Dobcross, Oldham	45
Garstang, T. W. H., M.R.C.S., Dobcross, Oldham	46
Johnson, Samuel, M.D., Stoke-upon-Trent	47
Williams, John, M.D., Swinton, near Manchester	48
Perry, Marten, M.D., Spalding	49
Fiddian, Alex. P., M.B., Cardiff	50
Davies, T. Arthur, M.B., Swansea	51
Latimer, H. A., M.R.C.S., Swansea	52
Farrer, Geo. A., M.R.C.S., Brighouse, Yorks.	53
Moorhead, T. H., M.D., Cootehill, co. Cavan	54
Allan, James, M.D., Leeds Infirmary	55
Booth, T. Mackenzie, M.B., Aberdeen	56
Stuart, J. A. Erskine, L.R.C.S. Ed., Dewsbury, Yorks.	57
Miles, J. F. Marshall, L.R.C.P., Dingle, co. Kerry	58
Burroughs, P. B., M.R.C.S., Weston-super-Mare	59
Walford, Walter G., M.D., 49, Finchley New Road, N.W.	60

43.—1. Prev. ill-health. 2. Severe protracted labour. Insan. conditions. 3. Miliaria on trunk and limbs. Sl. infl. sore throat. Mod. distn. of abd. Perimetritis. Sl. peritonitis. 4. Ut. enlarged and painful. 5. 2nd d. 6. Ab. 5d. 7. R. 8. Commenced with sudden rigors, fever, headache and vomiting on 2nd d. Patient v. restless and sl. delirious. Loch. suppressed and milk v. scanty.

Treatment, initial purgative, Dover's and grey powders, tonics, mild nourishing diet; Turp. stupes and poultices locally; evaporating lotion to head.

44.—2. Del. by midwife of putrid child. Breech presentation; much violence used; probable abrasions in vagina; sev. p. p. hæmorrhage. 3. Extreme distn. of abd. Uterine infln. 4. Ut. large and soft; os patulous. 5. —. 6. Sev. for 7 d. 7. R. 8. Called to case on 7th d. T. then 104°4, P. 130 v. weak, tongue brown and dry, features pinched, partial delirium, diarrhoea, cold perspsns.

Treatment, aconite, cinchona and pot. iod.; ut. carefully syringed thrice daily, much thick brown ropy discharge coming away each time, and T. falling 1 to 2 deg.; opium, quinine, liq. ferri perchlor. given subsequently.

45.—1. Dirty habits. No nurse. Intemperate. 2. Ch. stillborn. Forceps delivery. 3. Abd. distd.; pelvic infln. 4. Ut. large, soft, flabby. 5. 4 d. 6. 10 d. 7. D. 8. Fever severe from commencement.

46.—1. Dirty habits. No regular nurse. 2. Ch. stillborn. Instrumental deliv. 3. Abd. distd.; pelvic infln.; peritonitis. 4. Ut. large, soft, flabby. 5. 2 to 3 d. 6. 75 d. 7. D. 8. Syringing in this case not persistent. A large pelvic abscess burst externally above pubes. D. from exhaustion.

47.—1. Primipara; lacerated perineum. 3. Rash like scarlat on chest and abdomen. No sore throat. Abd. distd. 4. Ut. enlarged, painful. 5. Next d. 6. 7 d. 7. R. 8. Treatment, aconite, pot. chlor., cinchona; poultices; vag. injns.



48.—1. Primip. Difficult labour; forceps used; perinæum lacerated. 3. Great distn. of abd.; pelvic infln.; gen. peritonitis. 4. Ut. large and tender. 5. 24 h. 6. 5 d. 7. D. 8. Began with rigor, folld. by pain and tenderness of abdom. T. 103·5, P. 130. Loch. offensive. On last day P. 150, diarrhœa, vomg., delirium.

Treatment, ammonia, opiates, quinine, champagne; Turp. stupes; vag. injns.

49.—1. Primip. 33. Always great apprehension. 2. Measles epidemic at time. Placenta prævia. Version. Dead, decomposing fœtus. 3. Consid. distn. of abd. 4. Ut. somewhat large and tender. 5. Ab. 24 h. 6. 6 d. 7. D. 8. 2nd d. T. 103 M., 104 E., headache, and great thirst; some tenderness of abdom.; lochia scanty. 4 d. later, tympanites set in, no great tenderness; loch. purulent; T. 103·5 to 104·5. P. 100 to 130 weaker, delirium at intervals, app. good, less thirst.

Treatment, ergot after del.; Ol. Ricini on 3rd mornng.; Liq. ammon. acetat.; Belladonna to breasts. 4th d. Enema of turp. and castor oil; Magnes. sulph. and opium; Turp. stupes to abd.

50.—1. Tedious labour with impaction of head. Forceps used; bruising of maternal soft parts. 3. Abd. not distd. 4. Ut. subinvolted. 5. 4th d. 6. 14 d. 7. R. 8. Pain and soreness of vag. shortly after labour. No abdom. tenderness. Loch. never v. offensive. Fever and pain abated as free purulent discharge took place.

Treatment, quinine, benzoate of soda, opium, chloral; vag. injns.

51.—1. Highly nervous and hysterical. 3. Erythematous rash over both buttocks. Thrush-like sore throat, cured by glyc. boracis. Mod. distn. of abd. L. labium sloughed. 4. Ut. relaxed, tender. 5. 36 h. 6. 6 d. 7. D. 8. Primipara 40, labour slow. Forceps used; much bruising; consequent imperf. cleansing first 48 h. Diffused ecchymosis both buttocks. L. labium sloughed. Not much laceration of perinæum.

Death considered due to peritonitis and diffused inflammation caused by septic absorption.

Treatment, quinine, opium freely; great cleanliness; vag. injns. twice daily. Nurse had sore throat for 2 days.

52.—1. Amenorrhœa and hysteria for years previously. Puerp. fever prevalent about this time. Forceps used. 2. Had puerp. case, 1 m. previous, but had attended other cases without bad result. 3. Yellowish red erythematous blotches on extremities. 5. 27 h. 6. 48 h. 7. D. 8. Primip. 35. Forceps used. 2nd d. rigor and vomiting. 3rd d. T. 103·6, P. 120, much paroxysmal pain in abd.; loch. offensive. Calomel and opium given; frequent vaginal syringing. She became steadily worse. T. usually 104·2 and P. very small. On 3rd d. had much pain in arms and legs with rash as noted. D. on 4th d. Obstinate bilious vomiting throughout.

53.—1. Anxiety. 2. Primipara, 20. Difficult delivery by forceps; slight laceration. 3. Abd. distd. Sloughy condition of vag. which had diphtheritic appearance on 4th d. 4. Ut. not contracted. 5. Same d. 6. 5 d. 7. D. 8. Seen on 2nd d. T. 105, P. 160. tongue dry and glazed, had vomited once or twice; ut. large and soft, free discharge; no pain. Gave effervescing salines, brandy and soda, etc., with tepid sponging. 3rd d. T. 105·4 M., 105·5 E. sl. tenderness over abdom., less discharge. Sl. delirium, no sleep. Ordered, chloral draught, hot bran bag to abd., to continue salines. 4th d. T. 105·4 M., 105·2 E., P. 140, much delirium, no sleep. Abdom. pain much worse; no discharge. Ut. washed out; poultices with opium to abd.; mist. pot. chlor. c. acid hydrochlor. dil. given with chloral draught, and frequent brandy. 5th d. T. 105, P. 140. Washed out ut. Vagina had sloughing diphtheritic appearance. T. in Evg. 105·6, less pain, Ut. again washed out and chloral given. 6th d. T., 9 a.m., 106·4, P. 106. unconscious. D. same day.



54.—1. Severe labour 12 hrs. 3. Abd. distd.; pelvic infln. 4. Ut. enlarged, tender. 5. 4 h. 6. 4 d. 7. D. 8. *Æt.* 45. Second child; labour long and violent. First child, 1 y. previously. 6th h. after delivery, T. 102, no pain. 2nd d. T. 104 M., 102 E., no pain. loch. free. 3rd d. T. 104 M., 104·2 E. loch. ceased; ut. enlarged and tender. Aconite given without effect. 4th d. T. 104 M., 103 E. D. on 5th d. Some signs of pain for few hours before D. No rigor throughout. Treatment, aconite, quinine, opium. Leeches to abdomen.

55.—1. Prolonged labour. Injury to parts from cephalotripsy. Pelvis contracted and deformed. 2. No infection. 3. No rash. No sore throat. Abd. not dist. No local infl. in pelvis or elsewhere. 4. Ut. tender, slow in contracting. 5. 2nd d. 6. 4 w. 7. R. 8. During 1st w. T. 100–102. P. 102–132, taking saline mixt. with aconite. 9th d. T. 104·5, reduced by large doses quinine. 14th d. bloody disch. from ut. lasting 10 d., never foetid. T. from normal to 101. Patient primipara 21, with extreme ricketty deformity of pelvis. Labour long, and parts much bruised by necessary manipulations.

56.—2. Primip. *æt.* 30; large female twins. V. sev. labour; forceps used; perinæum torn. 3. Abd. not dist. 4. Ut. and loch. normal. 5. 14th d. 6. 6 d. 7. D. 8. Nurse neglected to syringe. 14th d. sev. rigors. T. 103·8. P. 140. R. 30 to 40. No abdom. pain; ut. normal. No chest symps. Treatment, Sodæ salicyl.; vag. injns. D. on 6th d.

57.—2. Primipara; narrow pelvis; protracted labour; delivery by forceps; laceration of vagina. 3. General pruriginous rash. Abd. distd., great tympanites. Cystitis. Phlegmasia Dolens. 4. Metritis. 5. 2 d. 6. Ab. 14 d. 7. R. 8. Delivery lasted 1 h. after applicn. of forceps. Vag. much torn. Ur. passed invol. from first. Severe cystitis at end of 14 d. Treatment, morphia suppos.; ice; vag. injns. Benzoate of soda, with daily washings of bladder; poultices with carbolic oil pledgets to vag. Patient about in 6 weeks.

58.—1. Prolonged labour. 3. Abd. distd.; pelvic infln. 4. Ut. enlarged and v. tender. 5. Few hrs. 6. About 10 d. 7. R. 8. Primip.; labour long. Version for arm presentn. Placenta removed by hand; much uterine pain. Next day T. 104. P. 140; abd. distd. and painful; retention of urine. Treatment, morphia, calomel; poultices to abd.; vag. injns.

59.—2. Primip. Forceps applied when head in pelvis; sl. laceration of perinæum; much bruising of internal parts. 3. Uniform bright scarlet flush on face neck and chest for some few hours; no desquamation. No sore throat. Abd. much distd. Infln. about uterus and espy. in L. ovarian region. Pneumonia L. base. 4. Ut. enlarged and v. tender. 5. 4th d. 6. 3 w. 7. R. 8. 4th d. Rigor, high fever, some delirium. Pelvic infln. then appeared, with tympanites. Lochia foul. Superficial sloughing of L. labium. 12th d. Pneumonia L. base; pelvic infln. rapidly disappearing. Treatment, salicylate of quinine; vag. injns; poultices.

60.—2. Whitlow on attendant's finger. 3. Abd. not distd. 4. Ut. enlarged. 5. 4th d. 6. 5 d. 7. R. 8. Primip.; labour tedious; long forceps used. 4th d. T. 104. P. 120; tongue furred; pain and tenderness over ut.; loch. scanty, offensive. Treatment, efferv. ammoniated salines; uterine injns.

## CLASS III.

## ORIGINATING IN, OR AFTER EXPOSURE TO CONTAGION.

## GROUP a.

(From Zymotic Diseases.)

## Section I.—Scarlatina.

NAME AND ADDRESS OF OBSERVER.	NO. OF RETURN.
Townsend, R. H., M.B., Queenstown, Ireland . . . . .	61
Westcott, W. Wynn, M.B., Dep. Coroner for Central Middlesex . . . . .	62
Coombe, R. Gorton, L.R.C.P., Tillingham, Essex . . . . .	63
Verrall, J. Jenner, F.R.C.P., Brighton . . . . .	64
Eakin, Samuel, M.D., Ballybay, co. Monaghan . . . . .	65
Wilkins, H. G. G., L.R.C.P., Ealing . . . . .	66
Roe, E. A. H., Surg.-Major A.M.D., 6, Whitehall Yard, S.W. . . . .	67
Donaldson, Ebenezer, Burt, near Londonderry . . . . .	68
Webb, Vere George, L.K.Q.C.P. Coleshill . . . . .	69
Jackson, Arthur, Shrewsbury . . . . .	70
Garner, J., M.R.C.S., Birmingham . . . . .	71
Purkiss, Arthur, M.D., 53, Kew Bridge Road . . . . .	72
Grosholz, F. H. V., M.K.Q.C.P., Aberdovey . . . . .	73
Stretton, John Lionel, M.R.C.S., Kidderminster . . . . .	74
Warner, Percy, L.R.C.P., Woodford, Essex . . . . .	75
Main, Walter, M.R.C.S., Clacton-on-Sea . . . . .	76
Reckless, Alfred, L.R.C.P., Shalesmoor, Sheffield . . . . .	77
Kyle, D. Hamilton, M.B., S. Andrews . . . . .	78
Rigby, Percy A., L.S.A., Lichfield . . . . .	79
Hastings, Horace C., M.R.C.S., East Dereham, Norfolk . . . . .	80
Lindsay, John, M.D., Lesmahagow, Lanarkshire . . . . .	81
Hadden, David H., L.R.C.S.I., Dublin . . . . .	82
Macphail, D., M.D., Whifflet . . . . .	83
Prowse, William, M.R.C.S.I., Clifton . . . . .	84
Hallett, Henry A., M.D. Aberd., Kimbolton, Hunts. . . . .	85
Muir, James S., L.S.A., Possilpark, near Glasgow . . . . .	86
Mitchell, Dugald, M.B., Renton, Dumbarton . . . . .	87
Rigby, Percy A., L.S.A., Lichfield . . . . .	88
Rigby, Percy A., L.S.A., Lichfield . . . . .	89
Macnab, James, L.R.C.S. Ed., Stirling, N.B. . . . .	90
Murphy, James, M.D., Sunderland . . . . .	91
Currie, D. W., M.B., Tillicoultry . . . . .	92
Lyddon, Richard, M.R.C.S., Ramsgate . . . . .	93
Ellis, H. D'Arcy, M.R.C.S., Brierley Hill, Staffordshire. . . . .	94
Raven, Thomas F., L.R.C.P., Broadstairs . . . . .	95
Stevens, W. Goldie, L.R.C.P., Edin., Renfrew . . . . .	96
Stevens, W. Goldie, L.R.C.P., Edin., Renfrew . . . . .	97

(The thirteen next cases are cases of Scarlatina.)

- 61.—2. Scarlat. prevalent in neighbourhood at time. Drainage doubtful. Enteric and diphth. had occurred in houses of same rango with same sewers. 3. Scarlat. rash over face, trunk, extremities; large patches of miliaria on upper arms and legs; desquamation followed. No sore throat. Abd. distd. tympanites. Great tenderness in ovarian regions and over whole abdom. 4. Ut. enlarged, tender. 5. 4th d. 6. 5 d. 7. R. 8. Primipara. Labour 2½ h. Placenta expressed 20 m. later. Labia contused. 4th d morn., had compl. of faintness during night. T. 101·8, P. 140, small

Flushed, anxious; tongue dry, brown; abd. tympanitic and v. tender, esp. over ut. and in each iliac region; ut. enlarged; lochia scanty and v. foetid. Ut. washed out; frequent vag. injns.; quinine and digitalis; stupes and poultices to abd.; milk, beef-tea, egg brandy. 5th d. T. 100, P. 112. Face, neck and chest covered with bright scarlet rash. No sore throat. Abd. tenderness less; lochia scanty, less foetid. 6th d. Rash v. abund. on trunk and upper arms. T. 100·2, P. 118. 7th d. Miliaria on arms; a few large purple spots on forearm, mixed with scarlat. rash. 8th d. rash fading. T. 100. 9th d. T. normal; rash nearly gone; tongue cleaning; desquamation on arms. Convalescent.

Child remained in room, but had no sign of scarlat.

62.—2. Mild scarlat. epidemic in district. 3. Scarlat. rash 5th day on face, chest, abdom. 8th d. Dusky blebs, size of sixpence on chest and limbs. Fauces congested; no memb. Abd. dist., tymp. 4. Ut. large, soft; loch. never v. offens. 5. 3rd d. 6. 5-d. 7. D. 8. Lab. natl. 3rd d. fever, hdche, sore throat. 4th d. Fever, hdche., scarlat. rash; throat sore and red. P. 120. No abdom. pain; loch. nat. Salines given. 5th d. Throat more sore; sl. abdom. pain; tymp.; ut. soft and large. 6th d. morn., more prostrate; diarrhoea; loch. less. Evening, rash discoloured, measles like. A dozen blebs of dusky serum on front of chest and limbs; sordes on teeth; tongue furred; great tymp. and abdom. pain. P. 135. Calomel and opium, brandy; turp. stupes. 7th d. weaker; delirious; less pain. P. 140. R. 20. Died suddenly; no P. M.

63.—1. V. bad sanitary conditions. 2. Scarlatina. Nurse had previously laid out 2 ch. dead of scarlat. 3. Scarlatinal rash over body. Throat sore. Abd. dist. Local infl. in pelvis. Jaundice. 4. Loch. profuse and foul. 5. 2 d. 6. 10 d. 7. D. 8. Primip.; forceps used. 3rd d. Rash. Sore throat. Metritis. Peritonitis. Jaundice on 6th day.

Treatment, calomel, opium, ergot, brandy; ice-bag; cold wet pack as required; Cond. injns. thrice daily. Child had scarlat.; also six children in next house, of whom 2 died.

64.—1. 1st confinement. Nervous. 2. Visited 2 wks. previously by relation who had been in a house with scarlat. 3. General scarlatinal rash, folld. by desquamation. Throat red and painful. Abd. distd. Infl. in L. inguinal region. 4. Ut. normal. 5. 3 d. 6. 45 d. 7. R. 8. Symptoms of scarlat. gone by 9th day. Next day T. 104·1. Symps. of L. inguinal parametritis gradually developed with great variation of Temp.

Treatment, large and small doses quinine. Poultices. Iodine. Salicin tried ineffectually.

65.—1. Mental depression. 2. Attended by sister with sore throat, whose servant had scarlat. at time. 3. General scarlat. rash, folld. by desquamation. Sl. redness of fauces and tonsils with pain. Abd. sl. distd., painful. 4. Ut. sl. large, tender. Loch. v. foetid. 5. 36 hrs. 6. 3 w. 7. R. 8. Severe scarlat. Patient v. ill some days. T. not above 104.

Treatment, bark, ammonia, and digitalis; tepid spongings; diaphoretics; gargles; poultices; vag. injns.

66.—1. Mother had had puerperal mania. 2. Measles epidemic in neighbourhood. Few cases scarlat. in same street. Was attending several cases at the time. 3. Scarlat. rash on trunk and legs on 2nd d. of fever, soon fading, followed by desquamation. Simple infl. of fauces. Sl. distn. of abd. Some infl. in R. side of pelvis. Boils in each axilla. 4. Ut. sl. enlarged, v. tender and painful. 5. 2nd d. 6. 2 mos. 7. R. 8. Primipara, 23. Lab. nat. 16 h. 2nd d. T. 101, pains in lower abd.; loch. scanty, not offens. 3rd d. rash appeared. Loch. offensive on 4th d. T. varied from 101 to 105. Occas. delirium.

Aconite, quinine, sodæ salicyl. given without effect on Temp. which was reduced by sodæ salicyl. with ammon. carb. Quinine and iron then given.



67.—2. No direct infection. 3. Rash resembling scarlat. over entire surface on 3rd d., lasting 4 d. Sl. redness of fauces. Abd. distl. towards close. 4. Ut. norm. till 3rd d. before D. 5. 15 h. 6. 15 d. 7. D. 8. 15 h. after del., vomiting and diarrhœa. 2nd d. pain and oppression in cardiac region. T. 105 in evening. Alarming convulsions on 5th d. Loch. scanty throughout, absent on 8th d. 12th d. ut. tender, some abd. distn. 14th d. loud systolic bruit at cardiac base. D. on 15th d.

68.—1. Grief. 2. Scarletina. 3. Typical scarlat rash on body and limbs. Tonsils, soft palate, uvula and pharynx, red and slightly swollen. Abd. not distd. 4. Ut. sl. subinvolution, tenderness at first. 5. 16 d. 6. 9 d. 7. R. 8. Lab. normal. Patient did excellently. Her eldest child died of severe scarlat. on 9th d. after her confinement. 16th d. fever, sore throat, rash. Milk arrested. 25th d. Temp. normal. Some pain in both hips on 26th d. No sequelæ.

Treatment. Expectant. Mist. ferri. perchlor.

69.—1. Nervous. Formerly intemperate. 2. Insan. conditions. Scarlat. in village. Had attended scarlat. on previous day. Nurse also had been staying in house where scarlat. present. 3. General dusky scarlatinal rash lasting 4 d. Sl. inflammation of fauces; no membrane. Abd. not distd. Pelvic infln. Arthritis in shoulders and knees (? scarlatinal rheum). 4. Ut. enlarged, tender. 5. 4th d. 6. 3 w. 7. R. 8. Allowed up by nurse for 1 h. on 4th d. Then rigor and pelvic pain. Opium; vag. injns.; turp. stupes to abd. On 6th d. rash appeared, with sore throat. Ut. symps. abated in a few days, but fever contd., patient being sl. delirious. Symps. abated at end of 3 w. On 7th d. husband who had been constantly in sick room developed diphtheria, with dense membrane and albuminuria; no rash. On 9th d. sister developed diphtheria and sl. erythem. eruption on chest. 21st d. mother, who had nursed all 3, had erysipelas, which began on face and later became general.

70.—2. Child with scarlat. in house at time. 3. Gen. scarlat. rash appeared 24 h. before delivery. Severe sore throat for 2 d. before rash. Abd. not distd. 4. Ut. and lochia normal. 5. 2 d. before. 6. 8 d. 7. R. 8. T. 103 evening after del. Varied from 105 to 103 for next 5 d., then rapidly fell. Treatment, salicylate of soda; vag. injns.

71.—2. Visit of a child with scarlat. 3. Gen. scarlat. rash. Mild sore throat. Mod. distn. of abd. Suppn. in pelvis. Abscesses on buttocks and thighs. 4. Ut. large, tender. 5. 5th d. 6. 3 w. 7. R. 8. 3rd confinement. This and prev. labours normal. 4th delivery 16 mos. later v. difficult by cephalotripsy. Prem. labour induced twice since. Pelvic brim much narrowed in antero-posterior direction.

72.—2. Scarlat. in neighbhd. 3. Scarlat. rash on neck, chest, arms. Sl. redness of fauces and tonsils; glands sl. enlarged. Abd. not dist. 4. Ut. norm. Loch. sl. offensive. 5. 3 d. 6. 5 d. 7. R. 8. Labour easy; after-pains severe. Fever soon subsided. Desquamation complete in 6 wks. Loch. soon healthy.

Treatment, salines, uterine injns. Pot. chlor. to throat. Tinct. cinchouæ co. and acid sulph. dil. during convalescence. Child nursed by mother throughout, but had not scarlat.

73.—2. Scarletina. 3. General red rash. Throat red, tonsils enlarged. Abd. distd. 4. Ut. enlarged and v. tender. 5. 2 d. 6. 2 d. 7. D. Case recorded in *Obstetrical Journal*, July, 1875, p. 237, and Sept., 1876, p. 379.

See also cases 17, 29, 47, 267, 289, 308.



74.—1. Severe p. p. hæmorrhage. 2. Scarlat. in room 14 d. prev. 3. Abd. distd. on 3rd d.; peri-utine infln. No rash. No sore throat. 4. Ut. v. tender. 5. Same day. 6. 7 d. 7. D. 8. Severe p. p. hæmorrhage. Child born before arrival. Blanched, almost pulseless. Ut. kneaded; placenta and clots at once expelled; then contracted well. In evg. T. above 100. P. 120, feeble. Became gradually worse and died on 7th d. T. reached 104·6.

Treatment, turp. internally; poultices; vag. injns; stimulants.

75.—2. Insan. conditions. Child with scarlat. in same bed until confinement. 3. No rash. Fauces congested; no exudation. Abd. sl. distd. 4. Ut. did not contract well, tender. Loch. offens. till 4th d. 5. 2 d. 6. 8 d. 7. R. 8. 2nd d. rigor, T. 104. 3rd d. T. 105, sl. sore throat. 4th to 8th d. T. falling. 9th d. rigor, T. 105. 10th d. T. normal and so remained.

Treatment, quinine; frequent vag. injns.

76.—2. Nursing children with scarlat. till 14 d. before del. 3. No rash. Sore throat. Abd. distd. 4. Ut. tender. 5. 3 d. 6. 6 d. 7. D. 8. 3rd d. rigor, very bad night. 4th d. sore throat and diarrhœa, sordes on teeth, muttering delirium at night. All symptoms increased and she died comatose on 6th d.

Treatment, ammon. carb.; garg. Pot. permang.; uterine injns.; stimulants. Good diet.

77.—2. Attended by assistant who had attended many cases malignant scarlat. Self had sore-throat, and T. 101 on day of delivery, and was in Fever Hospital for next three weeks. 3. Sl. raised papular rash on chest and abd. at end of 1st w.; no desquamation. Sl. sore-throat some days after rash. Abd. distd. Pelvic pain and tenderness. Gen. peritonitis. 4. Ut. large. Loch. normal. 5. At once. 6. 7 d. 7 D. 8. Labour easy. 2nd evening, after-pains v. severe. 3rd d. T. 103. Salicylate given. 4th d. T. 99·4. Salicyl. omitted. 5th d. T. 102·6. Salicyl. continued. 7th d. T. 101. Ut. washed out. Cinchonidine given. On 11th d. inflam. of both parotids. T. 102·2, P. 84. 14th d. Swellings subsided, no improvement, T. 103·6. 15th d. worse, T. 104. Dulness at bases of both lungs. Wet pack. D. on 16th d. Marked double pneumonia at close, its onset corresponding with decline of parotid swellings (? metastasis).

Cool packs gave great relief and had marked effect on Pulse and Temperature.

78.—2. Two cases scarlat. nursed by patient in same house for 14 d. before del. 3. No rash. Throat sl. inflamed. Abd. not distd. Pleuro-pneumonia. 4. Ut. norm. 5. 3 d. 6. 15 d. 7 R. 8. Sore-throat with sl. rigors 2 d. before del. 3rd d. after del., T. 103. Loch. became foetid. Cough, dyspnœa, pain in chest ensued on 4th or 5th d. Desquamation during convalescence.

Treatment, sedatives; vag. injns.

79.—2. Children convalescent from scarlat. at time. 3. No rash. No sore-throat. Great dist. of abd. Great tenderness over ovaries and whole abdom. 4. Ut. enlarged to umbilicus, extremely tender. 5. Ab. 5th d. 6. 19 d. 7 R. 8. Att. by midwife. Lab. lasted 3 d. Loch. offensive 5th d. Sent for on 14th d. T. then 103, great tenderness over whole abdom., esp. ut. and ovaries; lochia foul. Became grad. worse and seemed sinking of collapse. Posterior lip of os uteri and cervix sloughed. Gradually rallied. T. reached normal on 19th d.

Treatment, salicin, sodæ sulpho. carb. and bark; daily ut. injns.; nutritious diet and stimulants. Low form of ulceration later attacked fauces and tonsils; much sloughing with intense foetor.

80.—2. Slept with husband 14 d. prev. Husband developed scarlat. next day. 3. No rash. No sore throat. Sl. distn. of abd. Infln. in pelvis and in R. groin, and round to back. 4. Ut. normal. 5. 2 d. 6. Ab. 12 d. 7. R. 8. Some time previously had fallen and hurt R. groin, and had had pain there ever since. High fever, profuse perspn., and local pain in R. groin

on 2nd d. On 4th d. puffiness and boggy feeling extending round to back. Loch. seropurulent (? diffuse abscess opening into vag). Fever and puffiness soon disappeared but pain persisted in R. iliac region.

81.—2. Nursing child desquamating after scarlat. at time. 3. No rash. No sore throat. Abd. much distd.; probable peritonitis. 4. Ut. sl. large. 5. 4th d. 6. 3 to 4 d. 7. D. 8. 1st child; forceps used. Labour easy. 4th d. rigor. 5th d. high fever; abd. much swollen. Treatment, calomel and opium; poultices, etc.

82.—2. Scarlat. in house same time. 3. No rash. Throat congested and red; no exudation. Abd. little distd. 4. Ut. sl. tender. 5. 60 hrs. 6. 9 d. 7. R. 8. 3rd. d. M. T. 104. P. 120. Ev. T. 105. P. 124. 4th d. M. T. 104. P. 124. Ev. T. 105.5. P. 128. 5th d. M. T. 103. P. 124. Ev. T. 102. P. 114. 6th d. M. T. 101. P. 120. Ev. T. 100.8. P. 112. gradually falling to normal on 9th d. Treatment, diaphoretics, quinine, opium, pot. chlor.; stimulants; nutritious diet.

83.—2. Scarlatina. 2 ch. with scarlat. in back room at time. 1 ch. d. scarlat. 17 d. previously. 3. No rash. Throat painful; fauces injected. Abd. not dist.; no infl. in pelvis or elsewhere. 4. Ut. normal. 5. 72 h. 6. 12 h. 7. D. 8. Æt. 27. 5th ch. Labour normal. After-pains severe. Did well till evg. 3rd d., then v. restless, delirious through night, complaining of throat. 4th d. morn. exceedingly restless, rambling; vomiting. Bowels very loose; no abdom. pain. Skin cold and clammy. P. not felt at wrists. Ht. sounds weak, very rapid. Loch. ceased during night. Patient rapidly sank.

84.—2. Scarlatina. Doctor attending scarlat. at the time. 3. General suffusion; no distinct rash. No sore throat. Abd. not distended; no infl. in pelvis or elsewhere. 4. Uterus normal. 5. Within 24 hrs. 6. 3 d. 7. D. 8. High fever, temp. reaching 107.5°. Pulse 120–130, feeble. Lochia thin, not entirely suppressed. Child died on 6th day.

85.—2. 4 children ill with scarlat. at date of confinement. 3. No rash. No sore throat. Abd. dist., diffuse peritonitis. 5. 3rd d. 6. 3 d. 7. D. 8. Sudden fever (104°), distension of abdomen, violent delirium, exhaustion and death.

Morphia injected freely. Nourishment and brandy given often and in small quantities.

86.—1. Twins. Hand introduced within womb in delivery of 2nd child. 2. 1 ch. scarlat., other children measles, in same house 5 w. prev. Scarlat. and measles rife in district. Commenced attendance on bad case phlegmonous erysip. 3rd d. after del. Nurse attending daily till 3rd d. Death had occurred in nurse's practice 7 w. prev., but none since. 3. No rash. No sore throat. Abd. not distd. Limited infln. in lower L. lumbar region (? ovary). 4. Ut. soft and large, not tender. 5. 3rd d. 6. 7 d. 7. R. 8. 3rd d. T. 104.5, P. 140. Quinine and opium given. 5th d. T. 103, P. 130. 6th d. T. 102. Quin. and opium reduced. 9th d. T. 100.5. Quin. and opium again reduced. Localized pain low in L. lumbar region from 2nd to 9th day. P. very rapid at first, always soft. Loch. soon became scanty, milk also at first diminished.

Pain relieved by freq. sinapisms and fomentns. Vag. injns. used.

*(Doubtfully placed in this Group).*

87.—2. Scarlat. in same bed some 3 mos. prev. 3. No rash. No sore throat. Abd. not dist. 4. Ut. tender. Loch. ceased after 12 h. 5. 8 h. 6. 60 h. 7. D. 8. Primip. 8 h. after del., fever, headache, vomiting, and soon diarrhœa. 2nd d. T. 103, P. 140. Extreme restlessness and thirst. D. in 60 h.

Treatment, Dover's and grey powders, quinine and calomel; mustard poultices; vag. injns.

88.—2. Labour tedious. Epidemic of scarlat. in immediate neighbourhood. 3. No rash. No sore throat. Great distn. of abd. General peritonitis. Ut. much enlarged, nearly to umbilicus, v. tender. Loch. fœtid, slimy. 5. 3rd d. 6. 48 h. 7. D. 8. Lingering labour. Narrow pelvis; head long at brim; long forceps applied for ut. inertia. Placenta entire in 20 m. 3rd d. T. 101·3 M., 103·6 E. 4th d. T. 104·6 M. D. same d. P. running and rapid throughout; abd. tympanitic. Symps. of collapse. Treatment, salicylate and cinchona, with liberal ice. Brandy and good diet.

89.—2. Labour prolonged for 48 h.; long forceps used for ut. inertia. Placenta entire. The case occurred at end of epidemic of scarlat. 2 ch. d. scarlat. in next house shortly before. 3. No rash. No sore throat. Great dist. of abd.; pelvic infln. Great tenderness over whole abd., espy. ut. and ovaries. 4. Ut. much enlarged, nearly to umbilicus; lochia v. fœtid, scanty. 5. 5th d. 6. 16 d. 7. R. 8. 5th d. T. 101 M., 103·4 E. 9th d. T. 105·2 with great prostration and intense noise in ears; then slept 6 h. and gradually improved. Conval. on 16th d. Treatment, salicylate, sodæ sulphocarb., carbol. acid internally, then bark; ut. and vag. injns. daily; freq. turp. stupes to abdom. Nutritious diet with brandy.

90.—2. Scarlat. in neighbourhood. Patient had nursed two members of family with scarlat. 3 mos. prev. 3. Sl. dist. of abd.; sev. abdom. pain. Pneumonia L. lung ab. 15th d. 4. Ut. normal. 5. 4th d. 6. 8 d. 7. D. 8. 4th d. Sev. rigors. T. 102; pains in back and in sev. joints, resembling ac. rheumatism. Retention of urine throughout. Diaphoretics and turp. given. Pain in joints relieved, but T. high and abdom. pain with tendency to diarrh. till D. on 8th d. Child at breast till 5th d., had scarlat. 2 d. after mother's death.

91.—2. ? Chill on day before del. Attending several cases scarlat. and a case of erysip. same time. 3. Abd. distd. 4. Ut. large and tender. 5. 2 d. 6. 4 d. 7. D. 8. 2nd d. T. 102, violent pain in ut. No rigor. No vomiting. Ut. washed out. T. 105 before D. on 6th d. Treatment, quinine, opium; turp. stupes; vag. injns.

92.—2. Attending many cases, mild scarlat. and diphth. same time. Fever in 2 out of 7 puerp. cases attended. 3. No rash. No sore throat. Abd. distd.; pelvic infln. Peritonitis. 4. Ut. soft, tender. 5. 2nd d. 6. 3. 7. R. 8. Primip. slow labour; sl. lacern. of perinæum. 2nd d. rigor. 3rd d. T. 103·5 M., 104·6 E. tongue dry; abd. tynip. and tender; pain above pubes. Loch. and milk normal. 4th d. rigor. T. 105 M., 103 E. less pain. T. varying from 104 to 99 next 14 d. Normal after 18th d.

Treatment, aconite, quinine; poultices. Diet, milk and beef-tea.

93.—2. Scarlat. in neighbouring house at time. 3. No rash. No sore throat. Abd. normal. 4. Ut. normal. Loch. ceased 6th d. 5. 5th d. 6. 31 d. 7. D. 8. Labour easy. Placenta entire. 5th d. two rigors. 6th d. some vomiting; loch. ceased. 7th d. T. 101·5. P. 144. R. 48. 8th d. T. 103 M., 101 E. Rigor. Patient desponding and restless. Dry tongue, anorexia, thirst; skin acting freely. B. o. loose. During next week T. varying from 103·6 to 99, with 3 deg. diff. between M. and E. During 3rd w. some improvement; no rigors. T. and P. lower. At end of 3rd w. rigors returned. Pulse and resps. quick; tongue dry and tremulous; sordes on lips; profuse diarrhœa, muscular tremblings. D. 5 wks. after delivery. P. M. no infln., no typhoid ulcer. ut. flabby.

Treatment. 1st w., quinine and ergot. 2nd w., ferri. perchlor. 3rd w., large doses quinine. Local injns. Diet of beef-tea and milk, with 5 to 10 ounces brandy daily.



94.—2. Insan. condns. of house. Epidemic of scarlat. Had attended 20 cases scarlat. same day, but precautions taken. 3. Tenderness in pelvis. 4. Ut. hot and tender. 5. 5th d. 6. 10 d. 7. R. 8. Labour easy. Loch. healthy. Doing well till 5th d., then severe rigor, T. 104, P. 120. Quinine and aconite. Eveng. T. 100, P. 108. Cinchonism, medicine discontd. 6th d. T. 105, P. 130. Sodæ salicyl. Eveng. T. 102, P. 130. Condition much same next 4 d. No milk. Loch. slightly fœtid. 9th d. Ut. washed out; no clots. 10th d. T. and P. normal. No further urgent symps.

95.—2. Patient much exposed to scarlat. during prev. 5 mos., had nursed case of enteric in same house. Believed to have slept on mattress used by child convalesce. from scarlat. Insan. condns. 3. No rash. No sore throat. Abd. distd. Gen. peritonitis. Ut. large, tender. 5. At once. 6. 48 h. 7. D. 8. Primip. Ut. inertia; forceps used.

After birth of child, much tar-like fluid escaped from ut. Case serious from very beginning. In a few h. general peritonitis and metritis. Child d. 1 w lat-r of pyæmia.

Doctor's hand severely poisoned.

Of two nurses, one had had sore throat; the other a sharp attack of facial erysip.

96.—2. Attending case 115, confined on same day, and later proving fatal; going many times from one to the other and examining at each visit. Scarlatina in town at time. 3. Sl. distn. of abdom; liver congested. 4. Ut. large and tender. 5. 12 h. 6. 3 d. 7. D. 8. Seemed doing well after deliv. Delirium on morn of 2nd d., with severe abdom. pain, and constant bilious vomiting. D. on 3rd d.

Treatment, opium, bismuth, quinine; fomentns.; vag. injns.

97.—1. Floor of house inundated. Dusky and seeming ill 3 m. prev. 2. See note 2 in preceding case. 3. Abd. distd.; pelvic inflam. Probably congested liver. 5. At once. 6. 3 d. 7. D. 8. Labour slow; pain immediately after. Next d. delirium and vomiting.

Treatment, quinine, opium; fomentns.; vag. injns. Child died jaundiced on 2nd d.

## Section II.—Erysipelas.

NAME AND ADDRESS OF OBSERVER.	NO. OF RETURN.
Stalker, A. M., M.B., Dundee . . . . .	98
Nomi, William, M.D., Nova Scotia . . . . .	99
Lammiman, Cleland, F.R.C.S., Tunbridge Wells . . . . .	100
Platt, Wm. Henry, L.R.C.P., Kilburn, N.W. . . . .	101
Hollaund, Lucius, M.D., Newcastle-on-Tyne. . . . .	102
Hart, Francis T., L.K.Q.C.P., Much Wenlock, Salop . . . . .	103
Cook, Augustus H., M.R.C.S., Hampstead . . . . .	104
Raven, Thos. F., L.R.C.P., Broadstairs . . . . .	105
Thomson, W. Sinclair, 40, Ladbroke Grove, W. . . . .	106
Burman, C. Clark, L.R.C.P., Ed., Belford, Northumberland . . . . .	107
Rowbotham, H. C., M.R.C.S., Melbourne, Derbyshire . . . . .	108
Browning, George, M.R.C.S., Sheffield . . . . .	109
Platt, William H., L.R.C.P., Kilburn, N.W. . . . .	110
Platt, William H., L.R.C.P., Kilburn, N.W. . . . .	111
Bernard, Walter, M.R.C.S., Londonderry . . . . .	112
Hooker, Charles P., L.R.C.P., Cottishall, Norfolk . . . . .	113
Sutherland, Wm., L.F.P.S., Capheaton, Newcastle-on-Tyne . . . . .	114
Hardey, E. P., L.R.C.P., Sprink Bank, Hull . . . . .	115
Willet, Geo. G. D., M.R.C.S., Bristol . . . . .	116
Martin, Paulin, M.R.C.S., Abingdon, Berks . . . . .	117
Martin, Paulin, M.R.C.S., Abingdon, Berks . . . . .	118
Monteath, David, M.D., New Abbey, Dumfries, N.B. . . . .	119
Walker, H. Bownes, L.R.C.P., Lowestoft . . . . .	120



(The Six next Cases are cases of Erysipelas.)

98.—1. Weakness, overwork, anxiety. Attended by midwife. 2. Erysip. of R. leg, extending from small varicose ulcer 2 d. before del. Bad drainage. 3. Erysip. rash on R. leg spreading upwards to thigh, commencing 2 d. before labour as erythema with consid. œdema; later, diffused patches of subcut. infiltration. Inflamm. also in R. shoulder. 4. Ut. and lochia normal. 5. ? before labour. 6. 8 d. 7 D. 8. 3rd d. after del. T. 102, anorexia, malaise, and leg as noted. 5th d. T. 102, no sleep, very excited, tongue dry and glazed, the leg more red and serous effusion now becoming purulent. Ut. normal. Ordered opium pill, bromide draught; lead lotion to leg. 6th d. T. 101·2, face pinched, sleepless, much pain in R. shoulder. Ordered quinine and 6 oz. port wine daily. 7th d. T. 100·2. Tenderness, pain and swelling in R. shoulder. Leg looking quiet. B. constipated. Tinet. opii. at night. 8th d. Wandering, P. weak, no pain, inflamm. gone from leg. 9th d. Extensive œdema of R. upper arm; death.

99.—1. Erysipelatous condition of blood at time of delivery. Attending several cases enteric fever at time. 2. Hour glass contraction of ut. with retained placenta, removed by hand. 3. Eruption on lower limbs of fine pustules on inflamed base. Consid. distn. of abd. Vaginitis and urethritis. Large erysipelatous swellings on gastrocnemii and below elbows. 4. Ut. large and tender. 5. 48 h. 6. Ab. 9 d. 7 D. 8. Postpartum hæmorrhage. Adherent placenta, removed by hand. 2nd d. uterus swollen, hard and tender; lochia scanty. Calomel and opium given; poppy fomentns., light poultices, turp stupes to abd. 4th to 7th d. T. 103 to 105, P. 120 to 130, great thirst, muscular tremors. 7th d. large hard red very painful swellings on calves of legs and in muscles of lower arms, with intense pain. Mercurials given, then quinine, iron and stimulants; ut. washed out. About 7th d. a discharge of pus took place from external genitals. Death comatose on 10th d.

Infant died "in very erysipelatous condition" shortly after death of mother.

100.—2. Nursed by mother who had just recovered from erysip. of leg. 3. Erysip. rash from vulva down thigh on 11th d. Abd. distd. Pelvic infln. 4. Ut. enlarged, v. tender. 5. 3rd d. 6. 14 d. 7. R. Treatment, opium, quinine; vag. injns.

101.—2. Attending case of erysip. of scalp at time. Sanitary condns. not good. 3. Abd. distd. 4. Ut. enlarged. 5. 48 h. 6. 4 d. 7. D. 8. Ætat 40, primipara; labour prolonged, forceps used. Perinæum much torn, silver sutures inserted. Opium and vag. injns. given. Rigor 48 h. after del. T. 101·9. 3rd d T. 104. loch. foul and milk not secreted. 4th d. sutures removed for sl. erysipelatous blush about wound. Free aperient given with mixt. of sulphate soda and liq. ammon. acet. 5th d. abdom. v. tender and painful, severe sickness. Quinine and opium; frequent vag. injns. Restlessness and insomnia throughout. D. on 6th d.

See case 111.

102.—2. Erysip. in house 14 d. previous. Patient had washed linen of erysip. patient. Scarlet efflorescence general over body except face and chest, slightly raised, patchy round knees, varying much daily in colour, vividness, and extent, lasting 3 d.; no desquamation; no sore throat. Abdom. distd. later. Enteritis. No local infln. in pelvis. 4. Ut. normal. Lochia scanty. 5. 4-5 d. 6. About 14 d. 7. R. 8. Fever, vomiting, severe abdom. pain at commencement. T. variable, reaching 105. P. to 120. Tongue white, moist; apathy; sleeplessness. Loch. fœtid and scanty, gradually ceasing. Rash varied with temp. in place and extent; appeared for few hrs. over abdom. during slight relapse. Abdom. tender, tympanitic; spleen not tender. Case regarded as enteritis erythematoides.

Daily rectal and vag. injns. given. Poultices to abd. Bismuth and morphia, and later quinine. Diet, milk and soda water.

103.—1. Strumous constitution. 3. Erysipelatous rash on R. thigh with phlebitis R. femoral vein ending in abscess; probably pelvic infln. 4. Ut. tender, not enlarged. 5. 4 d. 6. 1 mo. 7. R. 8. Lab. nat. 4th d. rigor, fever. Abdom. never much distd. or painful. Occas. delirium. T. varying from 100 to 104; great depression. Loch. scanty and offensive. Milk suppressed. T. norm. 3rd w. Then crysip. and sl. congestion R. lung 2 d.

Treatment, small doses quinine; antiseptic vag. injns. Abscesses poulticed, then incised.

NOTE.—Erysipelatous rashes occurred also in cases 189, 215.

104.—2. Severe idiopathic erysipelas under same roof. 3. Abd. much distd. 5. 3rd d. 6. 2 d. 7. D. 8. Lab. easy. 3rd d. T. 103; ut. washed out. 4th d. T. 105, vomiting set in. Ut. washed out twice; large dose quinine twice daily. Patient unconscious 24 hours after commencement and so remained till D. on 5th d.

105.—1. Fatigue. Sea sickness. 2. Was nursing sister with erysip. Insan. conditions. 3. Abd. distd.; peritonitis; pelvic inflamm. Phlebitis. 4. Ut. normal. 5. At once. 6. Many w. 7. R. 8. *Ætat.* 30. Miscarr. at 2nd m. Severe hæmorrhage. Putrid placenta removed piecemeal, sev. d. later. Peritonitis and pelvic cellulitis followed. Ut. washed out with dilute tinct. iodi.

Treatment, opium and quinine; generous diet and free stimulants.

106.—1. Primipara, *Æt.* 20. 2. Erysip. in same house 5 mos. prev. 3. Slight sore throat. Abd. distd.; local inflam. in pelvis. Meningitis. 4. Lochia foul. 5. 4th d. 6. 10 d. 7. D. 8. In house for 2 w. before confinement.

Treatment, thoroughly antiseptic.

107.—1. ? Expos. to cold. 2. No direct infection. Had for some time attended case of erysip. but no vag. exam. made between delivery and onset of fever. 3. Abd. distd. 4. Ut. and lochia normal. 5. 8 d. 6. 9 d. 7. D. 8. Intense fever with copious sweatings; vomiting not v. severe; abd. sl. distd., no pain. Lochia normal. Marked delirium last 3 d. of life. T. 108.2 on day of death.

Quinine, digitalis, salicylic acid, opium, mineral acids, given without effect. The case gave rise to a slight epidemic; this was the only fatal case.

108.—1. Accoucheur had attended case erysip. same day, and had not washed hands since, was with patient some hours and made repeated examinations. 3. Abd. sl. distd. Loch. scanty and offensive 30 hrs. after del. 5. ? 12 h. 6. 2½ d. 7. R. 8. Lab. natural and easy. Rigor 10 min. after del. Placenta then removed by hand introduced into ut. Next d. frequent shivering with severe ut. pain. 3rd d. T. 102, P. 120, flushed, hot skin, tongue dry and furred; abd. distd. and sl. tympanitic; ut. enlarged, fixed and tender; lochia scanty, not offensive. Quinine given; opiate fomentns. to abd.; frequent ut. injections. Same evening, severe rigor, T. 105, P. 140, R. 40; sl. delirium. No phys. signs in chest. B. confined. Treatment continued, with milk, soda, and freq. brandy. 4th d. T. 103.4, P. 104, R. 32; severe pain in lower abd.; lochia scanty, green, offensive. Ordered aconite and ammon. carb.; freq. ut. injections. In evening, T. 105, v. delirious, less pain. 5th d. Had slept a little; very weak; profuse perspsns. T. 98, P. 104; no delirium; abdom. still tender; lochia less offensive. Taking well. Diet and stimulants increased. Ev. T. 100, P. 104. 6th d. Good night; feeling much better, T. normal; lochia normal; tongue moist. B. O. Hence steady progress. Up on 10th d.

- 109.—2. Had been attending case idiopathic erysip. 3. Abd. distd.; pelvic inflammation. 4. Ut. enlarged, tender. 5. 3 d. 6. 8 d. 7. D. 8. 3rd d. Rigors, T. 104, P. 114, pain over lower abdom. 6th d. Diarrhœa, insomnia, delirium. D. on 8th d.

Treatment, effervescing salines, ammonia and bark; vaginal douche; brandy, eggs, beef-tea, etc.

- 110.—1. Fall from dogcart during 7th month; uterine pain afterwards. 2. Accoucheur had case erysip. in his care. 3. No rash. Abd. dist. 4. Ut. dist. 5. 6 hrs. 6. 7 d. 7. D. 8. Labour rapid. Placenta entire. 6 hrs. after del., rigor, T. 102. 2nd d. T. 104·6, then varying from 102 to 104·5, till D. on 7th d. Loch. most offensive. Milk never appeared.

Treatment, scammony, large doses quinine and tinct. ferri sesquichlor; freq. vag. injns. weak sulphurous acid.

- 111.—2. Came from case 101, which later proved fatal puerp. fever. Insan. conditions. 3. Abd. distd. 4. Ut. enlarged. 5. 48 h. 6. 4 d. 7. D. 8. 2nd d. Rigor, T. 102; abdom. painful and tender; lochia foul; no milk. 3rd d. T. 104·9. 4th d. till D., T. 102 to 103.

Treatment, calomel, sulphite of soda, quinine, opium; frequent vag. injns.; moderate brandy.

- 112.—1. Strumous ophthalmia, and enlarged cervical glands during suckling in 2 prev. confinements. 2. Had nursed husband with facial erysip. 3. Red papular rash on back of forearms. No sore throat. Abdom. distd. 4. Ut. enlarged, lax, tender. 5. 54 h. 6. 6 d. 7. D. 8. 3rd d. Angiopleucitis commencing in L. leg and spreading up L. thigh and flank and along inner side of L. arm and forearm.

- 113.—2. Child with erysipelas in next room; free intercourse. 3. No rash. Abd. distd.; pelvic inflamm. 4. Ut. hot and tender; lochia offensive. 5. 36 h. 6. 3 d. 7. D. 8. 2nd d. Severe pain over abdom. and esp. over ut., with sickness and diarrhœa; profuse sanious offensive discharge. Delirium towards close; no rigors.

Treatment, ferri perchlor., free carbolic syringing. Child had erysip. of hand and wrist 1 w. later.

- 114.—2. Had incised and dressed a case of erisipelatous cellulitis on preceding day. 3. Abd. distd. Inflamm. in pelvis and abdom. 4. Ut. v. tender. 5. 3rd d. 6. 4 d. 7. D. 8. Lab. easy. 3rd d. severe rigor. Continuous rise of T. and P. till D. on 4th d.

Treatment, salines, diaphoretics; ut. injns.

(Attendant had another case of puerperal peritonitis 5 days later. *See* case 136.)

- 115.—1. Apprehension. Poor and ill-nourished. 2. Had nursed brother with erysip. in adjoining room until confinement. 3. Abd. distd. 4. Os patulous. 5. next d. 6. 1 w. 7. D. 8. Lab. natl. 2nd d. T. 100, P. 150, lochia normal. 3rd d. T. 101, much constipn. 4th d. Abd. distd., but no pain; no milk. 6th d. T. 104 M., 103·2 E. 2 doses Warburgh's tincture. 7th d. T. 102·8, delirious; lochia not offensive. Quinine given; ut. and vag. washed out. 8th d. Much flatulent dist. of abdom. Rapidly sank. T. 108 1 h. before D.

(*Doubtfully placed in this Group.*)

- 116.—2. Had seen case facial erysip. same day. 3. No rash. Abdom. not distd. 4. Ut. norm; lochia scanty. 5. 7 d. 6. 4 d. 7. R. 8. Primipara 27; labour easy. 7th d. rigor, T. 102·6, profuse perspsns., headache; loch. and milk scanty; breasts not painful. 8th d. T. 101·4; lochia absent. Milk increased; some pain in breasts. Convalescence gradual.

Treatment, mist. sennæ, opium, ammon. carb.; hot fomentns.; vag. injns. Case considered one of milk fever.



117.—2. Primipara; lingering labour; forceps used; perinæum lacerated; was attending patient's father for erysip. abscess till 2 d. prev. 3. Sl. pelvic inflam. ; paralysis of bladder for 2 w. 4. Ut. large and tender. 5. Ab. 72 h. 6. 14 d. 7. R. 8. 72 h. after deliv., rigors. T. 102, P. 130; tenderness over ut. Fever for 12 d. T. not above 102·3.

Treatment, salicylate and opium for 5 d. Catheter used. Bladder washed out with weak sol. quinine twice daily; vaginal injns.; free stimulants.

118.—2. Primipara; forceps used; consid. laceration of perinæum. Attending case facial erysip. same time. 3. Abdom. much distd.; tympanites; gen. peritonitis; cystitis. 4. Ut. large, tender. 5. 48 h. 6. 28 d. 7. R. 8. 48 h. after deliv., rigors; great pain in abdom., with much tympanites. T. from 101 M. to 104·4 E. Much pus in urine.

Treatment, sodæ salicyl., opium for 3 w. Catheter used, and bladder washed out with weak sol. quinine twice daily; vag. injns. Champagne freely. Large putrid clot passed in 4th w.; no further fever. Mammary abscess 5 w. after confinement.

119.—1. Sev. vomiting throughout gestation; conseq. debility. Mental depression and dread of confinement. 2. Had attended patient's father for cellulitis of face till 2 m. prev. Placenta adherent, removed by hand. 3. No rash; sl. catarrh of fauces. 4. Ut. sl. enlarged, tender. 5. At once. 6. 8 d. 7. D. 8. Primipara, 23; labour easy. Placenta adherent. Pulse weak and rapid from the first; repeated sweatings. T. not above 102. D. on 8th d.

Treatment, ammonia and bark; stimulants; uterine injns.

120.—1. Thrown from carriage 5 w. prev. Frequently complained of pain in R. groin during pregnancy. 2. Had attended cases of erysip. in charge of the nurses of this case within a few months. 3. Extreme distn. of abdom. 4. Ut. large and tender; sl. laceration of cervix. 5. 3rd d. 6. 7 d. 7. D. 8. Primipara, 31; delivery by forceps. 3rd d.  $\frac{1}{2}$  oz. castor oil given by nurse; violent purging followed in 15 m. Next d. T. 103. P. rapid and hard; intense abdom. tenderness and tympanites, with vomiting and purging. Milk and lochia v. scanty; lochia not offensive. Diarrhœa throughout. D. from exhaustion on 7th d.

Treatment, large doses tinct. ferri mur., quinine, opium, turpentine; ut. injections; starch and opium enemata; stimulants.

### Section III.—Enteric Fever.

NAME AND ADDRESS OF OBSERVER.	No. OF RETURN.
Bernard, Walter, F.K.Q.C.P., Londonderry . . . . .	121
Garraway, Edward, M.R.C.S., Faversham, Kent . . . . .	122
Taylor, James, L.R.C.P., Chester . . . . .	123

121.—1. Seven rapid confinements. Anxiety; bad nursing. 2. Children ill with enteric fever. 3. Dark coloured petechiæ here and there over body. Abd. distd. towards end. 4. Ut. relaxed, tender. 5. 2½ d. 6. 10½ d. 7. D. 8. Labour slow; post partum hæorrh.; perchloride of iron used. 4th d. rigor; T. 102·4, rapid pulse, anxious look, fretful. 5th d. rigor, T. 103, P. 136, R. quick; severe headache; uterus relaxed and tender; lochia free. 6th d. restless, T. 103; abd. tense and tender; vomiting. 7th d. T. 103, wandering; continued vomiting. 8th and 9th d. all symptoms worse. Death on 10th d.

Treatment, opium, belladonna, sulphocarbolate of soda, bark; stupes, poultices; ut. injns.; nourishing diet, stimulants.



122.—2. Mother nursing child with fatal enteric at time of delivery. 3. No rash. No sore throat. Abd. flatulent. 4. Ut. v. tender. 5. 3rd d. 6. 21st d. 7. R. 8. 3rd d. rigor, P. 150, abdom. pain; loch and milk suppressed. Sedatives given, and on 3rd d. patient apparently well. 9th d. recurrence of symps; herpes. about lips; tongue dry.

Treatment, sedatives, ammonia and supporting diet. Convalescent in 21 days.

123.—1. Nursing child with enteric until delivery. 2. No other infection. No enteric symps. 3. Abd. distended, ? pelvic inflamm. 4. Ut.—— 5. Before labour. 6. About 10 d. 7. D.

(Attendant had three other cases of puerperal peritonitis within following month; two fatal.)

---

#### Section IV.—Measles.

---

NAME AND ADDRESS OF OBSERVER.	NO. OF RETURN.
Mathers, Adam A. C., M.D., Coleraine, co. Derry . . . . .	124
Redpath, R. K. W., M.B., Edinburgh . . . . .	125
Tinley, Thomas, M.R.C.P., Edin., Whitby . . . . .	126

---

124.—2. Measles in house 1 w. prev. 3. Measles rash over whole body. Sore throat. Bronchial catarrh. 4. Ut. and lochia normal. 5. ? 12 h. 6. 3 d. 7. R. 8. Felt ill sev. d. before labour. Ch. stillborn. 12 h. after delivery, T. 101·5, P. 100, coryza, catarrh; measles rash; pelvic organs normal. 2nd d. T. 99·8, P. 100, rash fading. Speedy convalescence.

125.—1. Anaemia. 2. Primipara. Instrumental labour; lacerated perineum. Insan. conditions. Measles in house just before. Scarlat. on same staircase. 3. General raised rosy papules. Tonsils and uvula sl. inflamed; no membrane. Sl. distn. of abd. Sl. pain on pressure over pelvis. Bronchitis. 4. Ut. v. tender; lochia foul. 5. 2nd d. 6. 14 d. 7. R. 8. Primip. 20; small pelvis; lacerated perineum; child stillborn. Catheter used many days. Measles followed.

Treatment, quinine; ut. injns.

(Doubtful.)

126.—2. Husband developed measles on 3rd d. after del. Both had been exposed to the infection. 3. No rash. Abd. distd.; gen. peritonitis. 4. Ut. large, tender. 5. 2nd d. 6. 3 mos. 7. R. 8. High fever, and ut. tenderness on 2nd d. after del. Case lasted 3 mos. First ut. attacked, then one or both ovaries, then general peritonitis. Later, phlegmasia dolens.

Treatment varied with symps; diaphoretics, opiates, chloral, counter irritation, turp. poultices, enemata of assafoetida; later, quinine. This the first of 5 similar cases; all recovered.

---

#### Section V.—Rötheln.

---

NAME AND ADDRESS OF OBSERVER.	NO. OF RETURN.
Campbell, W. Macfie, M.D., Liverpool . . . . .	127
Ryan, J. M., M.B., Colchester . . . . .	128
Jardine, James, M.B., St. Helens . . . . .	129

---

- 127.—1. Apprehension. 3. Rash of Rötheln, universal, like scarlat. on thighs, like measles chest and face. Tonsils swollen, not ulcerated. Disch. of matter and blood from nose. Acute abscess of ears. Abd. distd. 4. Ut. normal; loch. never foetid. 5. 2 d. 6. 7 d. 7. R. 8. 3rd d. bad night, T. 104·5. Chloral c. aconite. 4th d. M. T. 104·6, E. T. 104·6. 5th d. M. T. 103, E. T. 103·2; rash appeared. Salicylate given. 6th d. M. T. 102·5, E. T. 102·8. 7th d. M. T. 101·5, E. T. 101·4. 9th d. T. and P. normal. Salicyl. stopped; quinine, acids given. Recovery now rapid.

Case regarded as uncomplicated but severe Rötheln in puerperal state.

(Doubtful.)

- 128.—1. Uncleanliness in house. 2. Several ch. had Rötheln at time 3. Slight tonsillitis. Abd. distd.; local inflamm. in pelvis and of right hip 4. Ut. large, tender. 5. About 10 d. 6. 2 w. 7. R. 8. Pelvic inflamm. with exacerbns. for many wks. Use of hip regained in 6 mos.

Treatment, febrifuges, sodæ sulphocarb.; poultices, hot fomentns.; vag injns.

- 129.—1. Anxiety. 2. Chill. Nursing 2 children with Rötheln. 3. One crop large measles-like rose spots on abdom. fading in a few days; no sore throat. Abd. distd. 4. Ut. normal. 5. ? 6. 17 d. 7. R. 8. Abortion at 6th mo. Wet through on preceding day. Next d. pain in left infra scap. region, followed by phys. signs of lobar pneumonia. 12th d. rash as above, lasting a few days; then enteric symptoms. T. 103, P. 130, tongue dry and coated; pain in abdom., espy. in R. iliac fossa; peasoup stools; retention of urine. From 17th d. gradual convalescence.

#### Section VI.—Variola.

NAME AND ADDRESS OF OBSERVER.	NO. OF RETURN.
Hunter, George, M.D., St. Catherine's, Linlithgow . . . . .	130
Raven, Thomas F., L.R.C.P., Broadstairs . . . . .	131

- 130.—1. Apprehension. Prev. confinement at 8th mo. Deliv. by forceps. 2. Had attended case mild variola 7 d. prev. and borrowed a book. Attended this patient wearing same coat and gloves as when reading book. 3. Rash on face, neck, chest; little on trunk or extrem. (i.) General scarlatiniform efflorescence, with fine yellow papules. (ii.) Distinct variolous pustules, followed by pitting. Marked distn. of abd.; pelvic inflamm. 4. Ut. much enlarged, full, heavy, tender. 5. 3rd d. 6. 30 d. 7. R. 8. 3rd d. rigor, T. 104·5, P. 120, pain and tenderness over ut. Grad. extension of pain to lower pelvic regions, with incr. in size of ut. and scanty, sl. foetid lochia. By end of 1st w., gen. peritonitis with much distn., folld. by effusion into peritoneal cavity and diarrhoea. Beginning of 2nd w. cellulitis in L. hypogastrium, and cystic collections of fluid in L. broad ligament. Grad. remission of symps. and tedious but good recovery.

Treatment, turp. epithems, then opium fomentns. and poultices; uterine and vag. injns.; quinine and salicylate.

- 131.—2. Variola. 3. Well-developed variolous pustules, general, chiefly on face; throat congested. 4. Ut. large and somewhat tender. 5. 2 d. 6. 8 d. 7. R. 8. Variolous eruption 2 d. prev. to delivery of healthy 7 mos. child. Child d. few days later with obscure symptoms of blood poisoning.

GROUP  $\beta$ .*(From other cases of puerperal septicæmia).*

NAME AND ADDRESS OF OBSERVER.	NO. OF RETURN.
Boulton, Percy, M.D., 6, Seymour Street, W.	132
Birt, George, M.B., Stourbridge	133
Birt, George, M.B., Stourbridge	134
Birt, George, M.B., Stourbridge	135
Sutherland, William, L.R.C.P., Capheaton, Newcastle-on-Tyne	136
Hurry, Jamieson B., M.B., St. Bartholomew's Hospital, E.C.	137
Palmer, Fredk. S., M.D., East Shéen	138
Bullock, J. E., M.D., 87, Ladbroke Grove, W.	139
Don, Arthur G., L.R.C.P., Sevenoaks, Kent	140
McVeagh, D., M.K.Q.C.P., Coventry	141
Fry, J. Farrant, L.R.C.P., Swansea	142
Gordon, E., M.R.C.S., Stockport	143
Webb, William, M.D., Wirksworth	144
Stevens, W. Goldie, L.R.C.P.Ed., Renfrew	145
Raven, Thos. F., L.R.C.P., Broadstairs	146
Terry, Henry G., M.R.C.S., Bath	147
Bullock, J. F., M.D., 87, Ladbroke Grove, W.	148
Hatchett, Joseph, M.D., Ashby-de-la-Zouch	149
Reid, John, M.B., Rochdale	150
Hooker, Charles P., L.R.C.P.Ed., Cottishall, Norfolk	151
Hunter, Wm. Lovell, M.D., Pudsey	152
Miller, J. W., M.D., Dundee	153
Caskie, Wm. Alexander, M.D., Largs, Ayrshire, N.B.	154
Neale, Arthur T., L.R.C.P., Newcastle-on-Tyne	155

132.—2. Attended by same midwife as case 164, but no vag. exam. made. Child born and placenta expelled before her arrival. 3. Abd. not distd. Pneumonia. 4. Ut. norm. Loch. scanty, not offensive. 5. 2 d. 6. 10 d. 7. D. 8. Seen on 3rd d.; then undoubted symps. of septicæmia, chiefly diarrhœa, septic pneumonia, prostration. D. 10 days after del.

Treatment, quinine, opium, acid sulph. dil.; ut. and vag. injns.; good diet and stimulants. Case much resembled acute tuberculosis.

Case 164 seen on June 26, 27, 28. D. on 29th. This case seen July 4th till D. on July 12th. Both attended by same midwife.

133.—1. Ill health. 2. This, with two succeeding cases, attended within 5 d. by same midwife. Scarlat. in neighbourhood. 3. Abd. distd.; infln. in pelvis and abd. and over liver. 4. Ut. tender; loch. sl. offensive. 5. 3 d. 6. 3 d. 7. D. 8. Began with rigors, diarrh., vomg.; vomg. increased till D. on 3rd d.

Treatment, salines, brandy.

134.—2. See note on case 133. 3. Abd. not distd. 4. Ut. large, tender. Loch. sl. offensive. 5. 3rd d. 6. 14 d. 7. R. 8. Primip. æt. 30; labour slow; some lacern of perinæum. 3rd d. rigor, hdche., T. 103°, P. 130. Prominent symps. hdche., insomnia, delirium. Abdom. symps. slight.

Treatment, salines, bark and acid, quinine, chloral and opium.

135.—2. See note on case 133. 3. Abd. not distd. 4. Ut. tender. Loch. sl. offensive. 5. 3 d. 6. 6 d. 7. R. 8. Labour slow; forceps used. Began with rigor.

Treatment, salines, bark and acid, quinine.



136.—2. Similar case 1 w. prev. Had incised erysipelatous cellulitis 1 d. before 1st case occurred. 3. Abd. distd.; pelvic infln. 4. Ut. v. tender, not large. 5. 3rd d. 6. 6 d. 7. D. 8. Lab. norm. 3rd d. T. 102, restless, tenderness over ut. Hot poultices to abd.; turp. and morphia internally. 6th d. all symps. worse. D. on 8th d.

137.—2. Attendant had visited puerp. case 4 d. prev. 3. Mod. distn. of abd. 4. Ut. tender, sl. fixed. 5. 21 h. 6. 7 d. 7. D. 8. T. above 103 for 4 d., never below 101·4. Salicylates and quinine given.

138.—1. Vomg. during pregnancy. Anæmia. 2. Assistant had attended case puerp. metritis 1 mo. prev. 3. Great distn. of abd. 4. Ut. large, boggy. 5. 3rd d. 6. 21 d. 7. R. 8. Tedious labour. Face presentn.; del. by long forceps. Some p. p. hæmorrh. treated by injn. of hot water. 3rd d. rigor, T. 103; abd. much distd.; severe abdom. pain. Later, constant diarrh., arrest of lochia and milk.

Treatment, aconite (with marked effect), opium; turp. stupes; ut. injns.; good diet, champagne.

139.—1. Privation. 2. Midwife had attended fatal puerp. case within 1 w. 7. Death 3 mos. after confinement.

P.M. notes. Recent pericarditis (2 wks.) No endocarditis. Recent small abscess in antr. mediastinum. Scanty recent lymph over both pleuræ. Acute bronchitis. Liver v. large, pale, soft and flabby. Spleen large and soft. Uterus normal. Sloughing cellulitis from L. side of ut., extending upwards to diaphragm and downwards below Poupart's lig. to open on surface. Old clot in L. fem. and iliac veins.

140.—2. Sev. similar cases about same time. 3. Consid. ascites. 4. Ut. — 5. 48 hrs. 6. 4 w. 7. R. 8. 3rd d. rigor, T. 103; severe pain and tenderness over whole abd.; loch. and milk suppd.; sickness, diarrhœa, dysuria.

Treatment, calomel, senna, opium; then opium, quinine and bismuth. Ascites ultimately disappeared. Compl. recovery.

141.—2. Had visited fatal puerperal case same day. 3. Abd. distd. Gen. peritonitis. 4. Ut. ? 5. 24 h. 6. 5 d. 7. D. 8. 2nd d. rigor, high fever, delirium; abd. distd., no pain. No milk. Loch. pale and offensive.

Treatment, opium; turp. fomentns.; vag. injns.; good diet, brandy.

142.—2. Attended by midwife having other puerperal cases at same time (2 fatal). 3. Abd. v. tympanitic. Enteritis. 4. Ut. and loch. normal. 5. 10 d. 6. 8 d. 7. D. 8. Labour normal. Did well till 9th d., when rigor. 10th d. T. 105, marked tympanites and abdom. tenderness. Mother died on 18th d. having discharging abscess in R. wrist and L. elbow, and another unbroken in R. foot. Child d. on 12th d. with abscesses in different parts.

143.—1. Mental anxiety. Unmarried. 2. Puerp. fever v. prevalent at time. Forceps used; previously used to a case wh. had proved septic, but immersed in hot water and carbolic. 3. No rash. Abd. dist.; infl. in pelvis; sloughing of vagina. 4. Ut. (p. m.) enlarged, gangrenous at placental site. 5. 3 d. 6. 10 d. 7. D. 8. Labour protracted; forceps used. Much distressed at birth of child. Child d. on 2nd d. overlain. 3rd d. slight rigor; loch. scanty; milk not secreted. T. 104. 4th d. peritonitis; pain in abdom.; severe diarrhœa; sloughing of vag. and labia; hdche.; violent delirium; coma. T. gradually rose to 107.

Treatment, quinine and opium; brandy; beef-tea, milk; ut. injns.



- 144.—1. Unmarried. Depression. 2. Consultant had another puerperal case. 3. Abd. distd.; pelvic infln. 4. Ut. enlarged, painful. 5. 4 d. 6. 4 d. 7. D. 8. Lab. easy. 4th d. rigor, with pain in lower abdom.; tympanites.  
Treatment, turp. stupes and hot fomentns.; nourishing diet, stimulants.
- 145.—1. Apprehension. Depression. 2. Had attended 2 fatal puerperal cases. 3. Great distn. of abdom.; peritonitis. 4. Ut. and loch. normal. 5. 36 h. 6. 4 d. 7. D. 8. 2nd d. rigor, hdche., abdom. pain. T. 105·5; peritonitic distension. D. on 4th d. Loch. normal throughout.  
Treatment, hyd. c. creta, quinine, opium; turp. stupes; vag. injns.
- 146.—2. Forceps used in severe puerperal case 6 weeks previously; had been washed in carbolic. 3. Abd. distd.; peritonitis; pelvic infln. 4. Ut. tender and painful. 5. At once. 6. 8 d. 7. D. 8. Primip. Forceps used for inertia. Retention of urine 2nd and following days. Lochia and milk gradually arrested. Vomiting, pain, diarrhoea. D. on 8th d.  
Opium, ammonia; vag. injns.; free stimulants.
- 147.—1. Similar case (not fatal) in same ward some wks. prev. 3. Confluent dusky red patches on abd. and hands, spreading to legs and thighs and becoming general. No sore-throat. Abd. not distd. 4. Ut. norm. 5. 3 d. 6. 7 d. 7. D. 8. 3rd d. rigors. T. 105·8 M., 103·4 E.; loch. scanty, foetid; little milk. 4th d. T. 105, delirious; tongue dry; disch. less. 5th d. T. 103. Hiccough and diarrhoea; confl. dusky red rash on abdn. and hands. 6th d. T. 104. Rash spreading in patches on thighs and fingers; lips and hands tremulous. 7th d. T. 105. Rash spreading. Less delirium. 8th d. T. 105, noisy delirium; increased tremor; disch. stopped; rash of brighter colour and general. 9th d. D. No P. M.  
Treatment, quinine, opium; alcohol; vag. injns.
- 148.—1. Overcrowding. 2. Probable puerperal infection thro. midwife. 3. Abd. much distd.; peritonitis. 4. Ut. normal. 5. 5th d. 6. 7 d. 7 R. 8. 4th d. diarrh. after a seidlitz powder, continuing till D. on 7th d. Fever. Abd. tense and tender. Loch scanty, never foul. Milk suppd. Obstinate sickness from first, becoming constant. Vomit at first bilious, on 6th d. faecal.  
Treatment, quinine, opium; ice, cold fluid diet; poultices to abdn.; vag. injns. No P. M.
- 149.—2. Delivered by midwife coming direct from house of fatal puerperal case. Scarlat. in same village. 3. No rash. No sore-throat. Abd. dist. 4. Ut. normal. 5. 4 d. 6. 8 d. 7. R. 8. 4th d. rigors for first few days. T. 103 to 104, P. 140 to 160. Drowsiness. Improvement gradual after 8th d.  
Treatment, frequent small doses liq. ferr. perchlor.; vag. injns.; stimulants.
- 150.—2. Infected by med. attendant. 3. No rash. No sore-throat. Abdom. distd.; pelvic inflamm. 4. Ut. —. 5. 28 h. 6. 17 d. 7 R. 8. Primipara. Forceps. Placenta removed and ut. well contracted. 2nd d. pains in belly; slight disch. of blood. Fomentations. 3rd d. T. 103·7; abd. tender; loch. muco-sanguinolent, not foul. 5th d. T. 100; loch. abundant and not foul. 7th d. T. 102·6; pain less. 8th d. T. 104·6; pain in belly.  
Treatment, opium, bark, acids, Dover's and grey powders.
- 151.—2. Had attended case 3 1 mo. earlier. 3. Sl. dist. of abd.; pelvic infln. 4. Ut. tender; loch. offensive. Milk free. 5. 3rd d. 6. 6 d. 7. D. 8. Del. easily with vectis. 2nd d. T. 100, rigor at night. 3rd d. T. 104. 5th d. restless, some delirium. Got worse daily and D. on 6th d. Nurse omitted to syringe till 3rd d.  
Treatment, bark and ammonia, bromide, chloral; vag. injns.  
(Infection possibly conveyed from case 3, attended one month prev.; 8 cases attended in interval without bad result).

(Doubtfully placed in this Group).

152.—2. Attending puerperal case same time. Woman nursing child with erysip. present at labour and in house daily. 3. Sl. distn. of abd.; pelvic infn. 4. Ut. soft and flabby, enlarged, tender. 5. 4th d. 6. 5 d. 7. D. 8. Lab. nat. 4th d. rigors. T. 103, P. 120 to 150. Great prostration from first. Severe continuous pain over lower abd. Loch. offensive on last day. Vomiting and diarrhoea from 3rd d. till D.

Treatment, opium; nutrient enemata; poultices, stupes; vag. injns.

153.—2. Examd. fatal puerp. cases 1 mo. prev. Attending erysip. 14 d. prev. and mild scarlat. at time. 3. Great distn. of abd.; pelvic infn. 4. Ut. tender. Loch. fetid. 5. 32 h. 6. 4 d. 7. D. 8. Long labour; forceps used high; sl. lacern. of perinæum. D. on 7th d. Much bilious vomiting throughout. Occas. delirium. No rigors, no diarrh. T. reached 105·6.

Treatment, cinchonidine and opium. Later, morphia suppos., nutrient enemata; free vag. injns; ice, champagne and stimulants.

154.—1. Apprehension. 2. Forceps used. P. p. hæmorrh.; placenta adherent; remd. by hand. Another fatal puerp. case same time. Attending phlegmon. erysip. 3. Abd. distd. 4. Ut. large, painful. 5. 4th d. 6. 5 d. 7. D. 8. Lab. severe. 5th d. feverish. 6th d. vomiting. Before death abdom. much distd. R. leg v. painful. R. ankle red œdematous.

Treatment, morphia; poultices; ice.

155.—2. Case next door attended by same midwife few mos. previously, who also died of puerperal fever. 3. No rash. No sore-throat. Abd. distd. No local infn. in pelvis, but cerebral infn. 4. Ut. large and tender. 5. 2nd d. 6. 5 d. 7. D. 8. 2nd d. fever, shivering, pain in uterus; loch. suppressed. Then general peritonitis, followed by wild delirium, rapid exhaustion and death.

Treatment, opium; poppy fomentns.; poultices, glycerine and belladonna to abdomen; vag. injns.

#### GROUP γ.

(From Septic Material.)

##### Section I.—Post-mortem Poison, or discharges from wounds.

NAME AND ADDRESS OF OBSERVER.	NO. OF RETURN.
Lowe, Walter G., M.D., Burton-on-Trent . . . . .	156
Illingworth, C. R., M.D., Clayton-le-Moors . . . . .	157
Alderson, Frederick H., M.D., Hammersmith . . . . .	158
Whittaker, W. M., M.B., Valentia, co. Kerry . . . . .	159
Whitwell, George G., M.B., Shrewsbury . . . . .	160
Prytherch, John, L.R.C.P., Liverpool . . . . .	161
Macphail, S. Rutherford, M.D., Carlisle . . . . .	162
Balding, D. B., F.R.C.S., Royston . . . . .	163

156.—1. Nervous. 2. Had handled wound discharging offensive pus. 3. No rash. Abd. not distd. 4. Ut. tender first 2 d. 5. 36 h. 6. 1 w. 7. R. 8. Labour normal; after-pains severe. 3rd d. T. 102, P. 120; restless and excited. 4th d. T. 101 M., 102 E.; pain in back and lower abdom. 6th d. T. normal.

Treatment, bromide, aconite, quinine; vag. injns.

157.—1. Unclean habits and surroundings. 2. Patient left unwashed for 48 h. after deliv. Was dressing more than one case of extensive burns. 3. Abd. slightly distd.; pelvic tenderness. 5. 3rd d. 6. 2 d. 7. R. 8. 3rd d. high fever; headache; abdom. tumid and tender; lochia foul. Sp. ammon., sodæ salicyl., and purgatives. 4th d. no change. Vag. injn. used. 5th d. no pain. Patient doing well.

158.—2. On morning of delivery had evacuated abscess in a fatal case of pyæmia. Had washed hands frequently before attending labour. ? chill on 2nd d. after del. 3. Abd. distd. Both lungs congested; fine pneumonic crepitation in postr. lobes. 4. Ut. hard and acutely tender. 5. 30 h. 6. 3 d. 7. D. 8. Lab. normal. Following evening, sl. shivering, restless; after-pains severe. Milk free. 2nd d. evening T. 100; abdom. distd.; severe continuous pain; ut. acutely sensitive; loch. normal. B. constipated; no sickness. Symps. severe from first. Temp. never above 102. Loch. sl. offensive towards close. Milk free throughout. No delirium. Treatment, calomel and opium, citrate of potash; turp. stupes, poultices with opium; vag. injns.; stimulants; nutritious diet.

(*Doubtful.*)

159.—1. Attending case of pyæmia at time. 2. Lacerated perinaeum. 3. Rash like scarlat. over left buttock and spreading up L. side of abdom. and chest, then over R. side and gradually fading. No sore-throat. Abdom. distd. Consid. uterine and gen. abdom. tenderness. 4. Ut. enlarged, tender. 5. 4 d. 6. 4 w. 7. R. 8. Began with rigors, abdom. pain and vomg.; lochia and milk suppressed. Symps. abated on app. of rash. Convalescence slow.

Treatment, quinine, opium; fomentns., poultices; vag. injns.

160.—2. Attending cases of erysip. Scarlat. in neighbourhood. Nursed by mother, who dressed foul nleer in same house. Insan condns. 3. Tympanites on 6th d. ? sl. effusion into peritoneum. 4. Ut. large, moveable, sl. tender; os patulous. 5. 8th d. 6. 23 d. 7. R. 8. Primipara; attd. by midwife. Hour glass contrn. of ut.; consid. p. p. hæmorrhage. Placenta adherent, removed by hand. 8th d. went downstairs. 9th d. sev. rigor, T. 106·4; severe headache; loch. scanty, sl. offensive; milk free. Quinine, ferri perchlor., and ut. injn. given. 9th d. T. 102·2 M., 105 E. Sodæ salicyl.; ut. injns. (carbolic). 10th d. deliriums in night; noises in head; some sickness; sl. carboloria; gave morphia and bismnth. 13th d. nterine bougies (iodoform and eucalyptus oil). 14th d. T. 104 M., 105·6 E.; all local treatment stopped; freq. tepid sponging; morphia; ol. terebinth. 18th d. T. rose to 106, looking v. ill, sl. muscular twitching, complg. of "stifling." 19th d. T. 98. 20th d. T. 105. T. normal on 30th d.

Patient had bronchitis for some days; never phys. signs of pneumonia. Double murmur heard in aortic regn. and soft systolic mitral. These disappeared as she improved.

161.—1. News of the death of a friend in childbed. 2. Primipara; long-continued labour. Had made post-mortem exam. of case of tetanns 12 h. prev. 3. Abdom. consid. distd.; pelvic inflamn. 4. Ut. large, tender. 5. 12 d. 6. 9 d. 7. R. 8. 14th d. after del. T. 104, P. 104. Resp. laboured; abdom. distd.; pain on sl. pressure over uterus or pelvis. Lochia absent. Milk free.

Treatment, pot. brom., tinct. lupuli; turp. stupes; free vag. syringing.

162.—1. Ill-health (jaundice) during whole of pregnancy. Acute excitement for 4 days, inducing premature labour between 7th and 8th mo. 2. Reporter had made 3 P. M. exams. in previous week. 3. Consid. distn. of abdom. 4. Ut. firm, but large. 5. 2nd d. 6. 9 d. 7. R. 8. Labour normal, but acute excitement continued, uncontrolled by sedatives. On 3rd evg. T. 102·6; lochia suppressed. 5th, 6th, and 7th d. muttering delirium, with T. 100 to 102. T. normal on 10th d. Abscesses formed later on scalp and dorsum of both hands; bed sores on sacrum. Long-continued mental depression followed. Convalescence slow, but complete.

Stimulants and nourishing diet given freely; no antipyretic.

163.—2. Attendant had made P. M. exam. on prev. day. 3. Great distn. of abdom.; local inflam. in pelvis and throughout abdom. 4. Ut. —. 5. 26 h. 6. 4 d. 7. D. 8. Primipara. Sev. rigor 26 h. after deliv., followed by metritis and peritonitis. D. on 4th d.

Treatment, calomel and opium; free stimulants.



Section II.—Insanitary Conditions.

NAME AND ADDRESS OF OBSERVER.	NO. OF RETURN.
Boulton, Percy, M.D., 6, Seymour St., W.	164
Wolstenholme, John H., M.R.C.S., Abergele, N. Wales	165
McGregor, James, L.R.C.P., Portsmouth	166
Luscombe, T. B., M.R.C.S., Teddington	167
Thompson, Arthur H., L.R.C.P., Great Ouseburn, Yorks	168
Maun, W. S., M.R.C.S., Edgbaston, Birmingham.	169
Kenny, J. B., M.K.Q.C.P., Killeshandra, co. Cavan	170
Rutherford, Thomas, M.B., Kelso, N.B.	171
Madden, Thos. More, F.R.C.S. Ed., Dublin	172
Beatty, W. J., L.R.C.P., Stockton-on-Tees	173
Nourse, W. E. C., F.R.C.S., Exeter	174
Hunter, Wm. Lovell, M.D., Pudsey, Leeds	175
Douty, Jas. Harrington, M.R.C.S., Powick, Worcester	176
Caley, Jas. William, L.R.C.P. Ed., Selby, Yorks	177
Mosse, Herbert R., Wandsworth Common, S.W.	178
Coates, W. Harrison, M.R.C.S., Hucknall Torkard, Notts	179
MacRae, John, M.D., Laggan, Inverness	180
Elliott, Christopher, M.D., Clifton	181
Wilks, George, M.B., Ashford, Kent	182
Wilks, George, M.B., Ashford, Kent	183
Hayes, Alfred A., L.K.Q.C.P., Cheltenham	184
Wake, Edward G., M.D., Dartmouth Park Hill, N.	185
Raven, Thos. F., L.R.C.P., Broadstairs	186
Raven, Thos. F., L.R.C.P., Broadstairs	187
Sheen, Alfred, M.D., Cardiff	188
Al'an, Francis J., M.D., 1, Dock St., London, E.	189
Sadler, Michael Thos., M.D., Barnsley, Yorks	190
Townsend, R. H., M.B., Queenstown, Ireland	191
Copley, William H., L.R.C.P., Wisbech, Cambs.	192
Barnes, Edgar G., M.D., Eye, Suffolk	193
Hill, Fredk. A., M.D., 76, Abbey Road, N.W.	194
Coombs, A. P., M.D., Castle Cary, Somerset	195
Coombs, A. P., M.D., Castle Cary, Somerset	196
Knight, Alex. A. H., M.D., Keswick, Cumberland	197
Cordes, A., M.D., Geneva	198
Moir, John W., M.C. Ed., S. Andrews, N.B.	199
Shaw, William, M.D., Maidstone	200
Heath, W. Lenton, F.R.C.S., 85, Gloucester Road, W.	201
Fiddian, Alex. P., M.B., Cardiff	202
Newman, William, M.D., Stamford	203
Cordes, A., M.D., Geneva	204
Macdonald, C. R., M.D., Beith, Ayrshire	205
Sheen, Alfred, M.D., Cardiff	206
Hamilton, A., L.R.C.P., Chester	207
Holroyde, J., Chatham	208
Watson, J. Adam, L.R.C.P., Chudleigh, Devon	209
Biggs, M. George, M.R.C.S., 93, Northcote Road	210
Balbirnie, John P., L.R.C.P., Staveley, Westmoreland	211
Gilmour, Thos. F., L.R.C.P. Ed., Glasgow	212

164.—2. Insan. conditions. 3. Abd. dist.; pelvic infln. 4. Ut. inflamed.  
 5. 2 d. 6. 3 d. 7. D. 8. 3rd d. labour easy and natural. Placenta  
 intact. Loch. norm.  
 Treatment, salicylic acid, calomel and opium; ut. and vag. injas.; poultices.



165.—1. Delicate. House cold and damp. Impure water. 3. No rash. No sore-throat. Abd. dist.; peritonitis. 4. Ut. slightly enlarged. 5. 3 d. 6. 2½ d. 7. D. 8. Primip. Labour easy. 2nd d. doing well; free disch. 3rd d. shivering; severe uterine pain. 4th d. evg. excited, much pain, P. 112, much flatus; some bilious vomiting. 5th d. bad night; lying on side. In evg. v. excited, P. 120, return of bilious vomiting. 6th d. morn. cold persps. Death.

Treatment, opium, calomel, aconite; turp. stupes to abd.; stimulants.

166.—2. Insanitary conditions. Patient ate quart of cockles 48 h. after delivery. 3. No rash. No sore-throat. Abd. dist. No local infl. in pelvis or elsewhere. 5. 4 d. 6. 3 w. 7. R. 8. Æt. 22, att. by midwife; labour normal. 4th d. enteric symptoms, vomiting, diarrhœa, pain in abd., especially in rt. iliac fossa; fever. No rash. T. normal at end of 3rd w. Convalescent in 6 w. Milk suppd. during fever, with consid. delirium. Lochia scanty and offensive, never suppressed.

Treatment, ammonia with bark, chloral and pot. brom.; vag. injns. Milk, mutton broth, small doses brandy.

167.—2. Reopening of cesspool in garden adjoining house on day after confinement. 3. Abd. distended; local infln. in pelvis, probably gen. peritonitis. 5. 2 d. 6. 5 d. 7. D. 8. 3rd d. rigors, vomiting. T. 100; lochia scanty. 4th d. rigor, sickness, T. 101, loch. offensive, abd. tender. 5th d. much same till evg., when T. 103, sickness, diarrhœa. 6th d. v. sick; loch. snppressed; delirious; abd. distended and v. tender, tympanites. 7th d. symps. progressing. 8th d. D. T. 105.

Treatment, purge, opium, sod. salicyl.; ut. and vag. injns.

168.—2. V. insanitary conditions. 3. Abd. distended. General peritonitis. 5. 2 d. 6. 5 d. 7. D. 8. On 3rd d. intense pain over nt., then over whole abd.; profuse sweatings, rigors, vomiting, prostration. T. to 105, P. to 130.

Quinine, perchloride of iron, opium; Condy injections; poultices.

169.—1. Placenta prævia with great loss. 2. Want of cleanliness and attention for 48 h. after del. 3. Abd. dist. Slight peritonitis. Ut. large, tender; loch. suppd. 5. 24 h. 6. 4 d. 7. R. 8. Placenta prævia; much loss. Found lying on soiled foul linen 48 h. after del. 3rd d. T. 102, P. 120, abd. tender; loch. ceased. Ut. inj. night and morning; quinine, generous diet, stimulants. 4th d. T. 103, P. 135, much hœche; abdom. tend. 6th d. convalescent.

170.—2. Very insanitary conditions. 3. Abd. distd. ?local infln. in lumbar regions. 4. Ut. large and inflamed. 5. 4th or 5th d. 6. 4 d. 7. R.

Treatment by calomel and opium, pot. chlor.; poultices and turpentine stupes to abd.; vag. injns. Occas. hypod. injn. of sulphuric æther. Good diet, stimulants.

171.—1. Temp. albuminuria 2 mos. prev. 2. Insanit. conditions. 5. 24 h. 6. 10 d. 7. R. 8. Primip. Rigor 24 h. after del. T. 104; loch. normal; abd. slightly dist.; no pain. T. 103 to 104 for next 7 d.; reached normal on 10th d.

Quinine; vag. injns. Good diet.

172.—1. Mental depression. 2. ?infection from lacerated cervix. Insanitary condn. of house. 3. Slight sore-throat. Abd. dist.; infl. in pelvis. 4. Ut. large, tender. 5. 5 d. 6. 3 wks. 7. R. 8. Labour slow; forceps used. Rigors on 4th d. 5th d. T. 105, P. 130, loch. and milk suppd.; slight tympan.; diarrhœa; profuse sweats; great depression; some delirium. Catheter used some days.

Treatment, quinine, opiates; warm antiseptic ut. injns.; poultices, turpentine. Generous diet and stimulants.

173.—1. Bad health. 2. Insan. conditions. Abd. dist.; genl. peritonitis. 4. Ut. large, tender. 5. 3rd d. 6. 8 d. 7. D. 8. 3rd d. evg., rigor, T. 103·2, abdom. tenderness, espy. on right side. 4th d. T. 105, increased tenderness; low delirium; all secretns. suppressed. Symps. much same till D. on 10th d. T. varying from 104·5 to 105·5.

Treatment, large doses quinine, calomel and opium; antiseptic injns.; turpentine stupes. Diet, frequent beef-tea, milk, brandy, eggs.

174.—2. Bad drainage. 4. Ut. norm.; lochia natural. 5. 4th or 5th d. 6. About 16 d. 7. R. 8. 4th or 5th d. rigors, P. rapid, feeble; anorexia; insomnia; debility.

Improvement began on closing untrapped sinkhole near door at foot of stairs.

175.—1. Ill-health. Depression. 2. Bad drainage. Scarlat. in house 12 mos. prev. House not disinfected. 3. Papular rash on chest. Slight redness of fauces; no memb. 4. Ut. soft, flabby. 5. 3rd d. 6. 6 d. 7. D. 8. Labour nat. 3rd d. rigor, diarrhœa, vomiting; little abdom. tend. T. varying 102 to 105, P. 130 to 160. Lochia not offens. D. on 7th d. Treatment, iodine injns. to ut. twice daily; diaphoretics, quinine; turpentine stupes.

176.—2. Insan. conditions. 3. Diphtheritic sore-throat. 5. 48 h. 6. 1 w. 7. R. 8. Placenta removed with diff., but whole. 3rd d. rigors, T. 103·4, P. quick, soft; intense hdche.; white patches on tonsils and fauces; some hypogastric tend.; disch. of blood clots.

Treatment, quinine, 20 gr. doses; vag. injns.; slop diet, brandy.

177.—2. Insan. conditions. Enteric fever in next house. 3. Abd. dist. Peritonitis. 5. 48 h. 6. 5 or 6 d. 7. D. 8. Primip. Labour rapid. 3rd d. fever, diarrhœa; great abdom. pain, P. feeble; lochia suppressed.

Treatment, quinine, opium; poultices. Ch. d. with erysip. in 4 wks.

178.—2. Bad drainage. 3. Sudam. rash on chest. Abd. dist. Enteritis. 4. Ut. norm. 5. 4th d. 6. 18 d. 7. R. 8. Primip. Labour nat. 4th d. rigors, fever; milk and loch. norm; no pain. 7th d. T. 100–102, hdche, lassitude, anorexia; loch. scanty, sl. offensive; milk stopped. Diarrh. and tymp. lasting 1 wk., checked by bismuth and opium. Symps. subsided at end of 3 w.

Treatment, quinine and ammonia. Later, iron, quinine and strychnia. Considered a case of gastro-enteric puerperal fever.

179.—1. Delicate health. Erysip. of head and face 6 mos. prev. 2. In. sanitary conditions. Scarlat. and diphth. in same row of houses within 2 mos. 3. No rash. No sore-throat. Abd. dist. 4. Ut. enlarged and tender. 5. 12 hrs. 6. 4 d. 7. D. 8. Attended by midwife; labour easy. In 12 hrs. diarrhœa, followed by abdom. distension and tenderness. Low muttering delirium. Lochia and milk suppressed.

Treatment, chalk and opium; free stimulants; turpentine stupes to abd.; vaginal injns.

180.—1. Ill-health, mental depression. 4th illegit. child. 2. Insan. conditions. 3. Abd. sl. dist. 4. Ut. normal. Loch. ceased gradually. 5. 6th d. 6. 12 d. 7. R. 8. 6th d. rigors, vomiting, diarrhœa. T. 102. P. 110, tendency to faint. Milk stopped. Diarrh. and vomiting continued some days.

Gave simple febrifuges at first, then calomel and opium; poultices. Milk diet stimulants.

181.—2. Damp house. Bad drainage. 3. Abd. dist.; pelvic and abdoml. infln. 4. Ut. tender and ?enlarged. 5. 2nd d. 6. 7 d. 7. R. 8. 3rd ch. Labour natl. 2nd d. rigors; rapid pulse; severe abdoml. pain. Later, diarrhœa. On 7th d. T. normal. Slight rise on evg. of 12th d. Then good recovery.

Treatment, frequent small doses tinct. aconiti. Later, ammon. carb., bark, quinine. Vag. injns. Milk diet.

182.—1. A presentiment of death. 2. Defective drainage. 3. No rash. No sore-throat. Intermittent distn. of abd. 4. Ut. not well contracted, os. patulous. 5. 5 hrs. 6. 23 d. 7. D. 8. Labour rapid. Placenta and membranes entire. 5 hrs. later, slight p. p. hæmorrh. 1st d. evg. restless, extreme apprehension, profuse perspsns.; loch. v. black. With much care, tonic and stimulant treatment, vag. injns., chloral at night, patient rallied, but T. and P. remained high. On 22nd d. while sitting in arm-chair sudden extreme faintness and dyspnœa. 23rd d. slight rally, but same evg. became unconscious, and died in a few minutes.

183.—1. Debility. 2. Defective drainage. Insan. conditions. 3. General vesicular miliary rash. Abd. not dist. Infln. in left knee and thigh. 4. Ut. norm. 5. 10 d. 6. 3 mos. 7. ? 8. 10th d. high fever, perspsns., swelling of left knee. These lasted some days. On 20th d. patient downstairs. 22nd d. return of fever and perspsns., with swelling and pain in left thigh. Abscess opened in left thigh on 42nd d.

Now in hospital for contraction of left knee-joint, and reported disease of toes of same foot.

184.—2. Insan. conditions. 3. Sl. distn. of abd. Metritis. Peritonitis. 4. Ut. dilated, v. painful. 5. 4th d. 6. 17 d. 7. R. 8. 4th d. sudden severe pain over lower part of abd. T. 103·6. P. rapid; tongue furred; severe hdche. In evng. T. 104, great abdom. tenderness; vag. hot and dry; loch. absent. Opium; poultices; vag. injns. ordered. Relapse on 11th d. Convalesc. on 18th d.

185.—1. Mental worry. 2. Variola in house 2 mos. before deliv. Insan. conditions. Uncleanliness after labour. 3. General miliary and papular rash. No sore-throat. Mod. distn. of abd.; local infln. over ut. and R. ovary. 4. Ut. v. tender. 5. ? 8th d. 6. 3 d. 7. R. 8. T. rose about 8th d. On 11th d. T. 104·5, profuse perspsns., violent headache, insomnia; sl. delirium; loch. scanty and fœtid. Milk scanty.

Treatment, aconite, veratrum viride, and pot. brom.; vag. injns. Later strychnine, perchloride of iron.

186.—2. ? impure drinking water. 3. No rash; no sore-throat. Abd. not distd.; pelvic inflmn. 4. Ut. large, hard, tender, painful. 5. 10 d. 6. 6 w. 7. R. 8. Attended by midwife. Rigors, fever, and uterine pain on 10th d. after deliv. Ut. remained large, tender, many weeks, with high temperature.

Convalescence slow.

187.—2. Insan. conditions. 3. Abd. distd.; pelvic infln. 4. Ut. large, tender, painful. 5. 4 d. 6. 10 d. 7. R. 8. 4th d. rigors, fever, pain in uterus. No milk. Loch. partially suppressed. Opium and antiseptic injns. used.

188.—1. Previous despondency. 2. Insan. conditions. 3. No rash. Diphtheritic sore-throat. Mod. distn. of abdn. 5. 3rd d. 6. 15 d. 7. D. 8. 5th d. T. 101, P. 120. 6th d. T. 101, P. 104. Hdche., constipn.; loch. offensive; tenderness of abdn. with some distension. Diphtheritic memb. on tonsils and fauces, finally extending to larynx. High fever till death on 20th d. Albumen  $\frac{1}{2}$ th on 12th d.

Treatment, quinine, aconite, digitalis, iron, pot. chlor., sodæ salicyl.



189.—1. Debility. No proper nursing. 2. Insan. condns. Uncleanliness after delivery. 3. Erysipelatous rash on R. side of face and neck; vesicles and bullæ on arms. Ulcerated throat. Abd. not distd.; sl. pelvic infln. 4. Metritis at first. 5. 5 d. 6. 2 mos. 7. R. 8. Æt. 23. 5th child. Much diarrhœa and weakness prev. to confinement; neglected during week following it. 4th d. great pain in both breasts. Axillary glands swollen and painful. Rapid sloughing of skin and cellular tissue of both mammae. T. 103·5 several days. Loch. profuse and fœtid. Improvement gradual. Later, erysipelas of L. side of head and face, with formation of bullæ. Treatment, quinine, acids, bismuth. Frequent nourishment, stimulants.

190.—2. Insan. conditions. 3. Abd. not distd. 4. Ut. and lochia normal. 5. 16th d. 6. 59 d. 7. R. 8. Did well till 16th d., then got up and complained of sickness and hdehe. 19th d. T. 102·8, P. 112, sl. diarrhœa. 21st d. T. 103·4, P. 118, no abdom. pain, tongue clean and moist. 30th to 35th d. T. from 98 M. to 103 E. From 40th d. daily rigors and sweatings, with T. 103 to 105, lasting 14 d., then gradual improvement. Slight thrombosis of R. femoral vein followed. Treatment, quinine, Warburg's tincture.

191.—2. Bad drainage. 3. No sore-throat. Abd. distd. Perimetritis. 4. Ut. enlarged, tender. 5. 2nd d. 6. 11 d. 7. R. 8. Lab. easy. 2nd d. evg. rigor; loch. stopped. 3rd d. T. 105·8, P. 142 M.; T. 103, P. 115 E., much ut. tenderness; loch. scanty, brown, fœtid; violent hdehe., sl. delirium. Slight rent of perinæum. Ordered quinine and digitalis; turp. stupes to abd.; ut. injns. Milk diet. 4th d. T. 99·4, tenderness and hdehe. gone. 5th, 6th, 7th days, improving. 8th d. sl. rigor. T. rose to 104·4 M., 101 E., loch. more abundant, not fœtid. Large dose quinine given. 9th and 10th d., fever remaining high. 11th d. T. 104·2, tongue parched; profuse perspsn. during the day. 13th d. T. and P. normal. No further rise of temperature.

192.—1. Insufficient food. Overcrowding. Debility. 2. Insan. conditions. 3. Raised papules scattered over abd. and extremities, espy. on arms and hands, becoming vesicles, then bullæ, oval, some as large as florins, full of purulent serum. Sl. dist. of abd. 1st wk. Infln. in large joints 3rd wk. 4. Ut. ? normal. 5. 3 d. 6. 4 w. 7. D. 8. 3rd d. rigor, fever, vomiting. 4th d. T. 103·2, P. 140, small, compressible. Resp. 45. Phys. signs of pneumonia of bases both lungs. Gave ammon. carb., bark, digitalis; turp. stupes; brandy. All symps. improved in 1st wk. Soon appeared convalescent. Later, rigors recurred, with diarrhœa and great pain in shoulders and knees. T. 103; albuminuria, hæmaturia, gradual coma. Death 1 mo. from onset.

193.—2. Searlat. in house 3 mos. prev. Neglected for 36 h. after confinement. 3. Sl. distn. of abd. 4. Ut. sl. enl. and tender. 5. 36 h. 6. 21 d. 7. R. 8. 2nd d. severe rigor, with profuse sweating daily for 1 wk., then occasional for 10 d. T. at close of rigor 105. Milk and loch. first partially suppressed, entirely at 10th d.; loch. offensive. Treatment, quinine; vag. injns.

194.—1. Dirt, drink, and insufficient food. Attending case acute peritonitis at time. 3. Abd. distd. 5. 2½ d. 6. 24 d. 7. D. 8. 3rd d. morn. T. 103·6, P. 120; eveng. T. 100, P. 120. No nurse. Room foul. 5th d. T. 101, P. 120, much exhausted. 8th d. loch. scanty, offensive; dull and stupid. 10th and 11th d. phys. signs of acute pneumonia entire R. lung. D. on 24th d.

Treatment, chloral; uterine and vag. injns. Good diet. Window of room was kept open day and night.



195.—2. Insan. conditions. 3. Abd. distd. Suppuration in shoulder and ext. malleolus. 4. Ut. norm. 5. 8 d. 6. 2 mos. 7. R. 8. Primip. Good labour. 8th d. fever, abd. swollen; loch. offensive. T. above 101 three to four weeks.

Treatment, quinine; vag. injns.

196.—1. Nervous. 2. V. insan. conditions. 3. No sore-throat. Abd. distd.; tenderness in pelvis. Meningitis. 4. Ut. large and tender. 5. 7 d. 6. 5 d. 7. D. 8. Became violently delirious on 9th d. and died unconscious 12 d. after del.

197.—2. Case of erysip. and of septicæmia 2 doors off. Insan. conditions. 3. Abd. distd.; pelvic influ. 4. Ut. v. tender; loch. normal. 5. 3rd d. 6. 9 d. 7. R. 8. T. from 101 to 106. Hdche., sickness, thirst.

Treatment, quinine, opium, digitalis.

198.—2. Insan. conditions. 3. Abd. not distd. 4. Ut. soft, large. 5. Same day. 6. 8 d. 7. R. 8. Forceps used in 1st and 2nd labours; third, a miscarriage; this the 4th confinement. Version for arm presentation. Fever lasted 8 d.

Treatment by aconite, quinine.

199.—1. Constipation. Small, badly drained house. 2. Abd. not distd. Sl. tenderness above L. groin. 4. Ut. sl. enlarged; lochia nat. 5. 5th d. 6. 16 d. 7. R. 8. Lab. easy; large child. Fever ranged from 101 to 105·5.

Treatment, quinine; warm enemata; vag. injns. Had similar fever after a previous confinement in a small badly drained house.

200.—1 P. p. hæmorrhage. 2. Bad drainage. 3. Throat sore; dusky red pharyngeal infln. Abd. not distd. Infln. in Douglas' pouch. 4. Ut. flabby. 5. 7 d. 6. Ab. 1 mo. 7. R. (with anæmia). 8. Some p. p. hæmorrh. in 3 prev. labours at term. 4 misc. (4 or 5 mos.). Consid. p. p. hæmorrhage. 8th d. sev. rigor, T. 104. For 14 d. T. 101 to 105. 12th d. pain in R. thigh and groin. Ut. and vag. v. tender and sensitive. No thorough exam. made. 19th d. much pus passed per vag. T. fell to 100·4, less pain, but much prostration. 10 d. later, another discharge of pus per vag., with fall of Temp. Convalescence slow.

Treatment, ammonia and bark chiefly, with free stimulants.

201.—2. Insan. conditions. 3. Gen. swelling and redness of fauces; no ulceration. Great dist. of abdn.; gen. pelvic tenderness, but no perimetritis or parametritis. 4. Ut. bulky, tender. Loch. copious, sanious. 6. 10 d. 7. R. 8. Illness commenced with chills, vomiting, diarrhœa, which became v. severe and almost constant. Abd. became much dist. and tympanitic, but not v. tender. T. 104 E., falling to 102 M. P. 120 to 150, feeble. Patient passed into typhoid condition, from which, however, she could always be aroused. On 6th d. was removed to a friend's house near, and within 24 h. there was marked improvement in all symptoms. T. fell rapidly, and diarrh. quickly ceased.

Stop diet, bismuth and small doses opium.

202.—2. Attending case of erysip. in previous week. Insan. conditions. 3. No rash. Abd. distd. at close. General peritonitis. 4. Ut. normal. 5. 16th d. 6. 14 d. 7. D. 8. Patient slowly convalescent until 16th d., then shivering, pain in abdomen. T. 101. 2 d. later diarrhœa, pain in micturition. These symps. continued till last day, when tympanites developed with uterine pain, prostration and high fever. D. on 30th d.

Treatment, salicylate of soda and morphia.

203.—2. Insan. conditions. 3. Infln. in pelvis and R. lung. 5. 7th d. 6. 3 w. 7. R. 8. Began by sev. rigor; pain in abd. and in R. side. T. 104, loch. foetid. Ut. mopped out; hot fomentns.; frequent vag. injns. Chest symps. some weeks. Phlebitis R. fem. vein. Treatment; salicylic acid, with much benefit.

204.—2. Insan. conditions. 3. Abd. uot distd. 4. Ut. relaxed; loch. offensive. 5. 2nd d. 6. 12 d. 7. R. 8. High fever every 2nd d. A true case of remitting fever during puerp. state.

*(Doubtfully placed in this Group).*

205.—1. Depression. Illegit. child. 2. Confined in low lodging-house. 3. Abd. dist. towards close. 4. Ut. normal. 5. 2 d. 6. 6 d. 7. D. 8. Labour natl. 3rd d. T. 102·5, P. 120, weak; hdcche.; slight bilious vomiting. B. n. O; lochia diminished; no milk. Ol. ricini given. On following days became worse. T. from 100 to 104, P. 125 to 135, weak and compressible; lochia suppressed. Last 2 d. delirium, tympanites, extreme depression. Treatment, digitalis, with large doses quinine; vag. injus; stimulants. D. on 7th d.

206.—1. ? Intemperance. 2. 2 ch. d. scarlat. 2 mos. prev. 3. Abd. not distl. Bad drainage; much sickness in house. 5. 3rd d. 6. 4 d. 7. D. 8. Labour uormal. 3rd d. sev. rigor. T. 105, great pain in abd.; loch. scanty; milk free (2 large doses quinine). Ev., T. 104·4, P. 144 (small doses quin. and vag. injns.). 4th d. T. 104·4; vomiting; Ev., T. 103·2, abdn. sore; some di-charge. 5th d. T. 102·2 M., 103 E., abd. tender; sickness, B.O. 4 (quin. each  $\frac{1}{2}$  h.). 6th d. T. 104·2, P. 144, R. 44, freq. vomg.; pain in abd.; anxious face; occasl. muttering delirium. Loch. scanty, dark, offensive. D. on 7th d.

207.—1. Tubercular diathesis. Albuminuria. Neuralgia. 2. Insan. conditions. 3. Soft palate and fauces congested. Abd. distd.; pelvic infln. 4. Ut. enlarged and tender. 5. Ab. 36 h. 6. 5 d. 7. D. 8. Labour easy. Hour-glass contraction of ut.; placenta removed by hand. Symps. of influenza, coryza and sore-throat same night. 2nd d. rigors, vomiting and diarrhœa. 3rd d. T. 103. Quinine and ut. injn. Improvement during next 2 d., then T. rose gradually to 105, with great tympanites and offensive diarrhœa. Injus. continued; carbol. acid, turpentine and stimulants internally. Patient gradually sank. D. on 6th d.

208.—1. Complete placenta prævia; much hæmorrhage. 2. Dead fœtus kept under bed 48 hours. 3. Abd. distd.; pelvic influ. 4. Ut. enlarged and tender. 5. 4th d. 6. 5 d. 7. D. 8. Primip. Lab. at 8th mo. Lab. completed quickly under chloroform. 4th d. several rigors. Loch. and milk soon ceased. Ut. enlarged. Abd. sl. tympanitic. Vomiting on 5th d. Convulsions and muttering delirium for 24 h. before D. on 5th d. Treatment, purgative, quinine and opium; poultices to abd; champagne, ice.

209.—1. 6th child in 6 years. 2. Insan. conditions. There had been epidemic of scarlat. in town. 3. No rash; no sore-throat. Sl. dist. of abd. 4. Loch. foetid, not suppd. 5. 3rd d. 6. 7½ d. 7. D. 8. Sev. rigor immediately after del. T. 100. In 6 h. T. normal. 3rd d. T. 102·8. Tenderness over abd., esp. in epigastrium. 4th d. T. 103, no abdom. tenderness, but gen. rheumatic pains and great thirst. V. drowsy. 5th d. T. 104 to 104·6, profuse diarrhœa; no pain. 6th d. T. 104 M., 103·4 E.; loch. v. foetid; diarrhœa persistent; semi-comatose, but taking well. 7th d. T. 103 M., 104 E., P. 150. Profuse diarrhœa in evening. D. on 8th d.

210.—1. Attended by midwife, who had badly ulcerated throat at time. In labour 12 hrs. 2. Insan. conditions. Constant illness in the house. 4. Aphthous state of mouth and throat. Bronchitis. Probably pyæmic inflamm. of hips and L. knee. 4 Ut. and lochia normal. Milk early suppd. 5. ? 5th d. 6. 3 w. 7. D. 8. First seen on 7th d. T. 103, P. 120, "light-headed." Large doses quinine given till cinchonism. T. again rose and diarrhœa set in, with aphthous mouth and throat. During last week of life T. subnormal, profuse persps., constant, severe pain in hips and finally in L. knee; no redness or swelling. Bronchitis 2 d. before death. Treatment, quinine, salicylate of soda. Good nourishment.

211.—1. Prev. ill-health. 2. Bad water supply. Sev. cases of mild diphth. sore-throat in village last 6 mos. Child taken ill with fever and sore-throat 1 w. later. 3. No sore-throat. Abd. not distd. 4. Ut. sl. tender. 5. 3rd d. 6. 10 d. 7. R. 8. Rigors and fever at commencement. Ut. v. tender. Loch. v. offensive. Abd. not dist. T. varying from 99 to 104. Constipn.

Treatment, opium, aconite, iodine; hot fomentns., poultices; vag. injns.

212.—1. Prev. ill-health. Bad nursing. 2. Forceps used. Insan. conditions. No direct infection. Searlat. prevalent in neighbhd. 5. Abd. dist. Infl. in pelvis, and of lungs and pleura. 4. Ut. large, flabby. 5. 3 d. 6. 7 d. 7. D. 8. Fever of irreg. remittent type, varying from norm. to 103; rose 2 h. before death to 106. Urine drawn off twice daily.  $\frac{1}{10}$  album. on 4th d. Lochia nat. No milk. Cough 4th d., with rapid breathing and shivering, rough ereps all over chest; no dulness.

Treatment, tepid sponging, turpentine stupes and aspersions; injns. Condy and carbolized glye. Ice *ad. lib.* Whiskey in frequent small doses. Niemeyer's antipyretic powders, grey powders.

#### GROUP δ.

(Fever commencing before Delivery, but no infection known.)

NAME AND ADDRESS OF OBSERVER	NO. OF RETURN.
Biddle, Cornelius, L.R.C.P., Merthyr Tydvil . . . . .	213
Ross, Roderick R., L.R.C.P., Stornoway, N.B. . . . .	214
Lloyd, R. R., M.R.C.S., St. Albans, Herts . . . . .	215
Garstang, T. W. H., M.R.C.S., Dobeross, Oldham . . . . .	216
Speirs, W. R., M.B., Haltwhistle, near Carlisle . . . . .	217
Cousellor, W. P., L.K.Q.C.P., Whalley, Lancashire . . . . .	218

213.—1. Anxiety and overwork. Had nursed husband with severe tonsillitis. 3 No sore-throat. Abdom. much distd.; inflam. in pelvis and in abdomen generally. 4. Ut. enlarged, inflamed. 5. 3 d. before del. 6. 11 d. 7. D. 8. Vomiting and severe pain in abdom. for 2 d. before delivery. Vomiting not much relieved by delivery. D. from exhaustion on 5th d.

214.—2. Pneumonia commencing 3 d. before delivery. 3. General blush over body. Sl. inflammatory sore-throat. Consid. distn. of abd.; influ. in pelvis. Pneumonia L. lung. 4. Ut. inflamed. 5. Before del. 6. 6 d. 7. D. 8. Pneumonia same side 6 y. previously. This attack v. severe. Extreme orthopnœa with delirium. D. by asphyxia on 3rd d. after del.

215.—1. Ac. rheum. 1875. Sore-throat (? diphtheritic) 48 h. before del. 3. Erysipelatous rash on face, secondary to glossitis. Inflammatory sore-throat with plastic exudation. Abdom. not distd. Phlegm. dolens in L. leg. 5. Few hours. 6. 7 d. 7. R. 8. Labour natural. 5 h. later throat worse, acute glossitis setting in. 7 h. later commencing facial erysip. 2nd d. glossitis better, sl. brouchitis. Taking fluids with stimulants well. 6th d. phlegm. dolens L. leg with erythema. Tinct. ferri. perchlor. given. 9th d. quinine and iron. 10th d. erythema less in thigh, spreading to leg and foot. 17th d. free discharge of pus from abscess in dorsum of foot. 19th d. throat still has greyish plastic lymph. Slough on dorsum of foot of similar appearance. 23rd d. abscess formed in middle of calf. Five mos. later patient able to walk, but not to straighten knee.



216.—3. Abd. distd. 4. Ut. not examd. 5. Concurrent. 6. 15 d. 7. D. 8. Fever began 3 d. before delivery. D. certainly due to pneumonia occurring on 8th d. after del.

217.—3. Abdom. not distd. Pleuropneumonia. 4. Ut. normal. 5. 1 d. before lab. 6. 7 d. 7. D. 8. On day before del. had sudden severe rigor and pain in side. When seen had fever, resp. short and rapid, dulcnss with friction over lower half R. lung. Ordered salines with aconite; poultices to chest. Next day, del. of stillborn child. Still pain in side, with cough, rapid resps., and some expectn. 3rd d. T. 104, great thirst; some delirium, no abdom. tenderness; lochia and milk normal; occas. diarrhœa. She gradually became worse and D. on 7th d. On 5th d. was called directly from this to another case, who on 4th d. after delivery developed all symps. of puerperal septicæmia and died on 11th d. Paid also a single visit to one of many cases attended by assistant. This case had rigor and T. 102 lasting sev. days, while all his other cases did well.

218.—1. Anæmic. Previous abortions. Acute mania 8 y. ago. 2. Recent enteric cases in neighbourhood. 3. No rash. No sore-throat. Abd. not distd. Obscure pelvic inflamm. 4. Ut. enlarged till expulsion of large clot on 3rd d. 5, 6, 7. See history below. 8. Abortion of fetus in 3rd month (early); entire ovum and maternal decidua expelled. T. 105. P. 150. Next d. T. and P. normal; lochia free. 4th d. rigor; ut. tender, a large clot expelled; frequent vomiting; poultices applied. 5th d. pain slight. great thirst. T. 103. 6th d. acute mania. 8th d. more composed. Frequent cough; much mucous expectn., loud râles. 10th d. P. 70, soft. T. normal; great pallor, restlessness; pupils widely dilated; tongue red and dry. 12th d. prostration, increasing till D. on 15th d.

Treatment, (1). Morphia and salines. (2). Bromide, chloral, digitalis. (3). Nitro mur. acid, pot. chlor. and tinct. cinchonæ. Brandy throughout.

## CLASS IV.

### COLD OR EXPOSURE.

NAME AND ADDRESS OF OBSERVER	NO. OF RETURN.
Williams, M. M., L.R.C.P., Chorley, Lancashire . . . . .	219
Mackintosh, M., M.B., Mortlake . . . . .	220
Garstang, T. W. H., M.R.C.S., Dobcross, Oldham . . . . .	221
Clarke, John C., M.R.C.S., Morley, Leeds . . . . .	222
Hughes, J. H., L.R.C.P., Ombersley, Droitwich . . . . .	223
Stuart, J. A. Erskine, L.R.C.S., Dewsbury, Yorks. . . . .	224
King, Henry W., M.D., Chester . . . . .	225
Boyce, Charles, M.B., Maidstone . . . . .	226
Andrews, Samuel, L.R.C.P., Basingstoke . . . . .	227
Blair, John, M.D., Shotts, Lanarks. . . . .	228
Mackenzie, Duncan J., M.D., Glossop, Manchester . . . . .	229
Bowkett, Thos. E., M.R.C.S., 145, East India Road . . . . .	230
Edwards, James, L.R.C.P., Liverpool . . . . .	231
Haining, William, M.D., Chester . . . . .	232
Allen, M. S., M.R.C.S., Dudley . . . . .	233
Rankin, Alexander, M.B., Glasgow . . . . .	234
Adcock, Harold, M.R.C.S., Middleton, Northamptonshire . . . . .	235

219.—1. Cold. 3. Herpes on lips. Abdom. not distd. 4. Ut. normal. 5. 16th d. 6. 5 d. 7. R. 8. Lab. nat. Took chill on 14th d. Next d. rigor, fever, herpes on lips. T. normal in a few days.



220.—1. Exhaustion. Bad nursing. 2. Exposed to draughts. 3. Abd. not distd. 4. Ut. normal. 5. 8th d. 6. 1 w. 7. R. 8. Sl. rigor, sev. headache on 8th d. T. 105·2, P. 130, sl. abdom. tenderness and pain across back. Lochia previously scanty now ceased. Milk secreted. Calomel and aconite given. 9th d. T. normal; no headache. 10th d. T. 103·2; tenderness over R. ovary; lochia reappeared. Quinine and ammon. citr. given. 11th d. T. 99, slept, and from this time made good recovery.

221.—2. Out of bed on 3rd d.; chill; followed by suppn. of lochia. 3. Abd. distd. Inflamm. in pelvis and elsewhere. 4. Ut. large, soft, flabby. 5. 4 d. 6. 56 d. 7. R. 8. Commenced with rigors, vomiting. T. 105 and lochia suppressed. T. once rose to 107. Convalescence slow. Quinine, salicylates and stimulants given, with free uterine syringing. There was an intercurrent attack of pleurisy.

222.—2. Severe chill on 4th d. 3. Abd. not distd.; pelvic inflamm. 4. Ut. enlarged, tender. 5. 4th d. 6. 7 d. 7. R. 8. Some p. p. hæmorrhage, sev. clots expelled. 4th d. took chill during night; shivering, severe uterine pain. T. 103·5. 5th d. pain increased in back and over ut. Lochia offensive. 8th d. T. normal. Treatment, salines, bromide, quinine; vag. injns.

223.—1. Mental anxiety. Debility. 3. Diphtheritic sore-throat; no rash. Abdom. much distd. 4. Ut. normal. 5. 7 d. 6. 9 d. 7. D. 8. Labour slow; forceps used for inertia. Went downstairs with insuff. clothing soon after delivery. 7th d. fever, sleeplessness. D. on 16th d. Treatment, salines, sedatives, stimulants.

224.—1. Delicate. 2. Forceps delivery. Chill from getting up too soon. 3. Abd. distd. 4. Ut.? 5. 12 d. 6. 7 d. 7. D. 8. Forceps delivery; no injury. Diarrhœa from first. Lochia suppressed. Treatment, antipyretics and stimulants.

225.—2. Chill. 3. No rash. Throat sl. congested. Abd. much distd. tender. 4. Ut.? Lochia foul. 5. 4th d. 6. 10 d. 7. R. 8. 4th d. took chill; shivering, feverish, excited. 6th d. T. 104·6, P. 124, headache, sickness, abdom. much distd. and tender; tongue dry; lochia dirty and v. offensive. Gave frequent vag. injns.; turp. stupes to abdom.; ice to head; acid. hydrocyan. dil. and quinine by mouth. Soda water and milk. Gradual complete recovery.

226.—1. Bronchial catarrh for 1 w. prev. 2. Went downstairs in night-dress on night of labour; sharp frost at time. 3. Abd. distd.; pelvic inflamm. 4. Ut. large, painful. Lochia scanty, not offensive. 5. 2 d. 6. 4 d. 7. D. 8. Gradual rise of T. to 105·6. Salicylates, quinine and aconite given; uterine injns. twice daily; brandy.

227.—2. Patient removed to fresh lodgings on 2nd d. after delivery. 3. No marked distn. of abdom.; some pelvic inflam. 4. Ut. tender and sl. enlarged. 5. 2½ d. 6. 10 d. 7. R. 8. Attended by midwife; labour easy. Moved from unsuitable lodgings on 2nd day. T. 104·5 20 h. after removal. 12 h. later pain in left side of pelvis. Pus in vagina on 4th d. Improvement rapid. Sodæ hyposulph. with ergot given.

228.—2. ? Chill. About house on day after delivery. 3. Abdom. slightly distd. 4. Ut. painful, tender. Lochia almost suppressed for 2 d. 5. 3rd d. 6. 3 d. 7. R. 8. 3rd d. fever, pain in pelvis; abdom. slightly distd.; loch. nearly suppressed. Treatment, pulv. ipecac. co., cold applics. T. normal in 4 d.

229.—1. Catarrh. 2. Inefficient nursing. 3. Ovaritis. Rheumatic pains. 4. Ut. norm. 5. 15th d. 6. 3 d. 7. R. 8. Headache and fever after confinement. Was up on 10th d. 15th d. fever. Quinine, vag. injns. given. 16th d. T. 104. Mercury and opium given. 17th d. better. T. fell gradually to normal.

230.—1. Gonorrhœa. Uncleanliness. 2. Exposed to cold. Slight laceration of perinæum. 5. 4th d. 6. 14 d. 7. D. 8. 4th d. rigor, T. 104; lochia foetid. Uterine inj. warm carbolic solution given, followed by slight epileptiform attack. Signs of pleurisy noted about 8th d. Left pleura tapped 8th d. and 1 pint of pus withdrawn. D. on 18th d.

231.—2. Symps. followed exposure to draught. Abdom. distd.; inflam. in pelvis. 4. Ut. v. painful; lochia checked. 5. 3rd d. 6. 4 d. 7. R. 8. Primipara; labour normal. Took chill on evg. 3rd d. Rigors. T. 103·4, P. 103, wiry; skin hot and dry; intense pain over ut.; lochia suddenly suppressed. Ol. ricini; vaginal injns.; turp. stupes to abdom. Dover's powder and quinine internally. 4th d. delirious during night. T. 102·4, P. 120, perspiring. Less abdom. pain; uterine pain continuing. Evening, T. 103·2, P. 130. Condy injections used; quinine and digitalis internally. 5th d. T. 100, P. 84, reg.; tenderness in L. iliac region only; lochia re-established. 6th d. T. and P. normal; no abdom. tenderness.

(Doubtful.)

232.—2. Chill. 3. Abdom. not distd. 4. Ut. normal; lochia scanty. 5. 7 d. 6. 4 d. 7. R. 8. Lab. normal. 2nd. half of placenta expelled by painful uterine contractions lasting 20 m. Evg. 7th d. milk absent both breasts. 8th d. T. 104·8, P. 120. 9th d. T. 102, P. 116. T. normal on 12th d.

Treatment, saline purgatives, sodæ salicyl., aconite, ferri perchlor.

233.—2. Insan. conditions. Cold room on 4th d. after delivery. 3. Sl. distn. of abdom. 4. Ut. enlarged and tender. Lochia and milk normal. 5. 4th d. 6. 4 d. 7. R. 8. 4th d. rigors. T. 130, P. 130. Sl. variation night and morning. Sudden improvement on 8th d.

Treatment, ammonia and bark, opium, brandy.

234.—2. Scarlat. in sister's house near by, 1 m. prev. Sat at open window on 4th d. after del. 3. No rash; no sore-throat. Abdom. distd.; some local inflam. in L. inguinal region. 4. Ut. normal. 5. 4 d. 6. 14 d. 7. R. 8. 4th d. sev. rigor; great pain in L. iliac region. T. 104 to 101 first 14 d., P. rapid throughout; pain persistent. A distinct swelling over L. ovary some days later; no sign of abscess per vaginam. Milk and lochia absent at end of 1st. w.; no return.

Treatment, quinine and morphia, acid hydrocyan. dil., bismuth. Nutritious diet; brandy.

235.—2. Primipara; very lingering labour. Up too early and exposed to cold and draughts. 3. Sl. distn. of abdom. Inflam. about uterus. 4. Ut. enlarged, tender. 5. 10th d. 6. 14 d. 7. R. 8. Attack began with severe rigor, feeling of sickness, thirst. Lochia, at first profuse, became scanty and v. offensive. 2nd d. T. 103, flushed and feeling sick, very restless, lower abdom. swollen and tender. Improvement gradual.

Treatment, opium, poultices; uterine and vag. injections.

## CLASS V.

## SHOCK OR EMOTION.

NAME AND ADDRESS OF OBSERVER.	NO. OF RETURN.
Jamieson, James, M.D., Edinburgh . . . . .	236
Ross, Roderick R., L.R.C.P., Stornoway, N.B. . . . .	237
Raven, Thos. F., L.R.C.P., Broadstairs . . . . .	238
Westby, George, M.K.Q.C.P., Liverpool . . . . .	239
St. George, George, M.K.Q.C.P., co. Antrim, Ireland . . . . .	240
Pocock, F. Ernest, M.D., North Kensington, W. . . . .	241

**236.**—1. Starvation and ill treatment during pregnancy. Sudden death of a brother in house 3 days after del. 3. Throat sl. congested towards end. Abd. not distd. 4. Ut. soft. 5. 5 d. 6. 14 d. 7. D. 8. Easy labour. 3rd d. brother died after 1 d. of illness. Fever on 5th d. 104 to 105, fell to 101 at end of week, but in 4 d. again rose to 105. D. on 9th d. Treatment, large doses quinine; ergotine suppos.; ut. and vag. injns.

**237.**—2. Up on 6th d. Ran to suddenly screaming child and was at once seized with flushing, headache and sickness. 3. V. sl. distn. of abdom. Breasts inflamed and painful. 4. Ut. normal. 5. 6th d. 6. 4 d. 7. R. 8. High fever, severe headache with great delirium and insomnia; both lochia and milk checked. No abdom. symptoms. Treatment, initial purge, opiates, bromide; evap. lotion to head and sinapisms to nape of neck; mustard foot-baths; fomentns. to breasts. Symptoms subsided on 4th d.

**238.**—2. Mental shock. 3. Abd. m. distd.; peritonitis; pelvic inflam. 4. Ut. large, tender, painful. 5. 4 d. 6. 3 d. 7. D. 8. Was in extreme fear of her nurse. Rigors, vomiting and diarrhoea on 5th d. after del. Uterus first inflamed; later, peritonitis.

**239.**—1. One brother an imbecile; a sister in asylum for 1 year after her first confinement. 3. Abd. not distd. 4. Ut. normal. 5. 2nd d. 6. 11 d. 7. R. 8. Extreme excitement, with T. ranging to 105·4, P. to 170, without any apparent cause. Treatment, quinine and opium.

**240.**—1. Nervous shock. 3. Abd. much distd.; pelvic inflam. 4. Ut. large, v. tender. 5. 8 d. 6. 4 w. 7. R. 8. Primipara, 40. labour slow; forceps used. Chill on 8th d. On 9th d. rigor; lochia ceased. 10th d. fever, delirium; abdom. tenderness; turpentine internally. Turpentine stupes; vag. (iodine) injns. Recovery rapid.

**241.**—1. Feeble health during pregnancy. 3. Abd. not distd. Inflam. of mammae. 4. Ut. norm. 5. 5 d. 6. 11 d. 7. R. 8. Doing well till 5th d., when frightened during night. 6th d. T. 103, P. 130–140. R. 32. Trembling. Breasts hard and knotty. Milk suppressed. Treatment, acid salicyl., quinine, iron; vag. injns.; poultices and fomns. to breasts.



CLASS VI.

CAUSE NOT ASSIGNED.

NAME AND ADDRESS OF OBSERVER.	NO. OF RETURN.
Macleod, John B., M.D., Dundee . . . . .	242
Weston, E. J. Darby, M.R.C.S., Handsworth . . . . .	243
Hurd-Wood, J., M.D., Leatherhead, Surrey . . . . .	244
Lawson, S., L.F.P.S., Brierfield . . . . .	245
Bernard, Walter, F.K.Q.C.P., Londonderry . . . . .	246
Fletcher, H. B., L.R.C.S., Sheffield . . . . .	247
Atkinson, R., M.R.C.S., Ripponden, Halifax . . . . .	248
Newman, William, M.D., Stamford . . . . .	249
Newman, William, M.D., Stamford . . . . .	250
Cox, William J., M.R.C.S., Chippenham . . . . .	251
Carageorgiades, S. G., M.D., Limassol, Cyprus . . . . .	252
Wright, Wm. Henry, L.K.Q.C.P., Derby . . . . .	253
Stalker, A. M., M.B., Dundee . . . . .	254
Cook, Augustus H., M.R.C.S., Hampstead . . . . .	255
Cook, Augustus H., M.R.C.S., Hampstead . . . . .	256
O'Callaghan, R. T. A., L.R.C.P., Bagenalstown, co. Carlow . . . . .	257
Fiddian, Alexander P., M.B., Cardiff . . . . .	258
Latimer, H. A., M.R.C.S., Swansea . . . . .	259
Fry, J. Farrant, L.R.C.P., Swansea . . . . .	260
Williams, Edward, M.R.C.S., Aberayron, Cardigan . . . . .	261
Donovan, William, L.R.C.P., Whitwick, Leicester . . . . .	262
Johnson, Samuel, M.D., Stoke-upon-Trent . . . . .	263
Mackay, John C., L.R.C.P., St. Columb, Cornwall . . . . .	264
Raven, Thos. F., L.R.C.P., Broadstairs . . . . .	265
Nettle, William, M.R.C.S., Liskeard . . . . .	266
Prideaux, Engledne, L.R.C.P., Wellington, Somerset . . . . .	267
Greenwood, Major, jun., L.R.C.P., Dalston, E. . . . .	268
Ladell, W. J. Simpson, L.F.P.S., 27, Canonbury Road, W. . . . .	269
Barnes, Henry, M.D., Carlisle . . . . .	270
Manser, F., M.R.C.S., Tunbridge Wells . . . . .	271
Stevens, Fredk. G., L.R.C.P., Bristol . . . . .	272
Gason, John, F.K.Q.C.P., Rome . . . . .	273
Gason, John, F.K.Q.C.P., Rome . . . . .	274
Arthur, James, L.R.C.P., Ferry Hill, Durham . . . . .	275
Williams, John, M.D., Swinton, Manchester . . . . .	276
King, Henry W., M.D., Chester . . . . .	277
Dickie, Thomas, L.R.C.P.Ed., Loanhead, Edinburgh . . . . .	278
Emerson, John J., L.K.Q.C.P., Kirkwall, Orkney . . . . .	279
Jones, James Thomas, M.R.C.S., Hornsea, Hull . . . . .	280
Frew, William, M.B.Ed., Galston, Ayrshire . . . . .	281
Beecham, Henry Jas., M.D., Ipswich . . . . .	282
O'Connell, Patrick, M.D., Sioux City, Iowa, U.S.A. . . . .	283
Hunter, W. L., M.D., Pudsey, Leeds . . . . .	284
Wood, Richard, M.D., Bromsgrove, Worcester . . . . .	285
Nesfield, Stephen, M.D., Manchester . . . . .	286
Ross, Roderick R., L.R.C.P., Stornoway, N.B. . . . .	287
Hetherington, George A., M.D., St. John's, New Brunswick . . . . .	288
Lawson, Samuel S., L.F.P.S., Brierfield, Burnley . . . . .	289
Lawson, Samuel S., L.F.P.S., Brierfield, Burnley . . . . .	290
Lawson, Samuel S., L.F.P.S., Brierfield, Burnley . . . . .	291
Hardie, David, M.B., Forres, N.B. . . . .	292
Hardie, David, M.B., Forres, N.B. . . . .	293
Hardie, David, M.B., Forres, N.B. . . . .	294
Palmer, J. Foster, L.R.C.P., 8, Royal Avenue, S.W. . . . .	295
Garstang, T. W. H., M.R.C.S., Dobcross, Oldham . . . . .	296



## 178 APPENDIX OF RETURNS ON FEVER IN THE

NAME AND ADDRESS OF OBSERVER.	NO. OF RETURN.
Garstang, T. W. H., M.R.C.S., Dobcross, Oldham . . . . .	297
Garstang, T. W. H., M.R.C.S., Dobcross, Oldham . . . . .	298
Garstang, T. W. H., M.R.C.S., Dobcross, Oldham . . . . .	299
Garstang, T. W. H., M.R.C.S., Dobcross, Oldham . . . . .	300
Tinker, Frederick H., L.R.C.P., Hyde . . . . .	301
Freer, William, M.D., Waringstown, co. Down . . . . .	302
Sheen, Alfred, M.D., Cardiff . . . . .	303
Sheen, Alfred, M.D., Cardiff . . . . .	304
Sheen, Alfred, M.D., Cardiff . . . . .	305
Evans, Thomas, M.D., New Quay, Cardigan . . . . .	306
Woodward, Martin, M.R.C.S., Pershore, Worcestershire . . . . .	307
Crichton, George, M.B., Twickenham . . . . .	308
Bullock, J. E., M.D., 87, Ladbroke Grove, W. . . . .	309
Crowe, J. W., M.D., Worcester . . . . .	310
Denby, T. Curtis, M.R.C.S., Bradford, Yorks . . . . .	311
Thomas, A. Garrod, M.D., Newport . . . . .	312
Chestnutt, J., L.R.C.P., Howden, East Yorkshire . . . . .	313
Macnab, James, F.R.C.S., Stirling, N.B. . . . .	314
Campbell, J., M.B., Ebchester, Newcastle-on-Tyne . . . . .	315
Stewart, John, L.R.C.P., Newport, Fife, N.B. . . . .	316
Beverley, W. H., M.R.C.S., Scarborough . . . . .	317
Townsend, R. H., M.B., Queenstown, Ireland . . . . .	318
Pinkerton, Charles, M.D., Southport . . . . .	319
Kinkad, Professor R. J., M.D., Queen's College, Galway . . . . .	320
Bridger, John, M.R.C.S., Cottenham, Cambs. . . . .	321
Hill, T., M.D., Islington, N. . . . .	322
Raven, Thos. F., L.R.C.P., Broadstairs . . . . .	323
Raven, Thos. F., L.R.C.P., Broadstairs . . . . .	324
Raven, Thos. F., L.R.C.P., Broadstairs . . . . .	325
Raven, Thos. F., L.R.C.P., Broadstairs . . . . .	326
Miller, J. W., M.D., Dundee . . . . .	327
Leigh, Thos. Drake, M.R.C.S., Liverpool . . . . .	328
Smith, P. Caldwell, M.B., Motherwell, N.B. . . . .	329
Meyers, Herbert H., L.R.C.P., Finsbury Park . . . . .	330
Biggs, M. G., M.R.C.S., Wandsworth Common . . . . .	331
Greenwood, Major, jun., L.R.C.P., Dalston, E. . . . .	332
Farrar, Joseph, L.R.C.P., Morecambe . . . . .	333
Wilbe, Richard H., M.D., 21, Finchley Road, N.W. . . . .	334
Smyth, Spencer T., M.D., Forest Hill, S.E. . . . .	335
Balding, Mortimer, M.B., St. Albans, Herts . . . . .	336
Bain, William, L.R.C.P., Manchester . . . . .	337
Walters, George B., M.D., Stonehouse, Gloucester . . . . .	338
Donovan, William, L.R.C.P., Whitwell, Leicester . . . . .	339
Weld, Chas. Humphrey, M.R.C.S., Hawkhurst, Kent . . . . .	340
Davies, E. J., M.B., Liverpool . . . . .	341
Davies, Francis J., F.R.C.S., Newport, Monmouthshire . . . . .	342
Sydney, Henry, M.B., Hounslow . . . . .	343
Green, Thos. Beaufoy, M.R.C.S., Kendal . . . . .	344
Reid, John, M.B., Rochdale . . . . .	345
Sadler, Michael Thos., M.D., Barnsley . . . . .	346
Gillespie, Franklin, Surg.-Major, A.M.D., Aldershot . . . . .	347
Ball, Edwin G., M.B. Ed., Birmingham . . . . .	348
Richardson, W. Brown, M.R.C.S., Blackpool . . . . .	349
Sloan, John, M.B., Bramley, Leeds . . . . .	350
Stuart, J. A. Erskine, L.R.C.S., Staincliffe, Dewsbury . . . . .	351
Havell, C. Graham, L.R.C.P., Felixstow, Ipswich . . . . .	352
Dale, Ridley, M.D., Sunderland . . . . .	353
Harper, Joseph, L.R.C.P., Barnstaple . . . . .	354

242.—1. Sl. metritis 14 d. after prev. labour. Pneum. of R. apex with tuberc. deposit. Apprehension. 3. Sl. distn. of abd. 4. Ut. sl. tender. 5. 30 h. 6. 4 d. 7. D. 8. Lab. nat. 2nd d. feverish and restless, sl. delirium. 3rd d. T. 103. Aconite in small doses. T. 101 E. 4th d. T. 99, loch. sl. foetid. Vag. injns. ordered. 5th d. stomach irritable. Ordered bismuth and hydrocyan. acid, champagne, ice; with subcut. injn. of morphia. 6th d. persistent vomiting; pain across stomach; abd. not distd.; no ut. pain. T. 99. 7th d. no sleep, vomiting persistent. D. in evening.

243.—2. ? erysip., not probable. 4. Ut. norm. Loch. slightly offensive. 5. 4 d. 6. 5 d. 7. R. 8. Same eveng. T. 104. No other symps. 2nd d. T. 103·5 M., 104 E. 3rd d. T. 102 M., 103 E. 4th d. T. 102 M., 102 E. 5th d. T. 101 M. 6th d. T. 100·5.  
Treatment, frequent vag. injns. ; cnemata ; small doses quinine.

244.—1. Primipara. 3. Great dist. of abd. 4. Ut. large, v. tender. Loch. abs. 5. 48 h. 6. 10 d. 7. D. 8. Placenta and memb. entire. No laceration. Loch. norm. first 3 d. Milk never appeared. Fever high throughout. T. 105·6 on 11th d. On 10th d. T. fell, surface cold and clammy; comatose; constant diarrhœa. D. on 12th d.  
Treatment, quinine, Warburgh's tincture, champagne, brandy.

245.—1. Despondency. Apprehension. 3. Abd. sl. distd. 4. Ut. large and sl. tender; loch. scanty. 5. 2nd d. 6. 4 d. 7. R. 8. 2nd d. T. 102·3; loch. scanty. 3rd d. T. 105, P. 100. 4th d. T. 106, P. 108. 5th d. T. 103, P. 95; hence rapidly recovered.  
Treatment, salines, bromide, chloral.

246.—1. Feeble health. Anxiety. 3. Abd. distd.; peritonitis; infln. about ut. 4. Ut. not well contd. 5. 4th d. 6. 3 d. 7. D. 8. Lab. normal. 4th d. morn. T. 102·8, P. 144; pain and tenderness over ut.; vomiting. In eveng. T. 101·4; abd. dist. and very tender; vomg.; respns. quick and laboured. 5th d. less pain, no vomiting; breathing improved. D. on 6th d.  
Treatment, aconite, opium, belladonna, quinine; stupes, and poultices; vaginal injns. ; nutrient enemata; stimulants.

247.—1. Prev. ill-health. 2. Scarlat. in district at time. 3. Sl. distn. of abd.; pelvic infln. Pneumonia. 4. Ut. large, soft, tender. 5. 2nd d. 6. 5 d. 7. D. 8. 3rd d. even. T. 104; great pain in iliac and hypog. regions; loch. scanty, v. offensive. Signs of pneumonia. Steadily got worse and died comatose on 5th d.  
Treatment, opium, ammonia and bark, quinine, sodæ salicyl.; vag. injns. Brandy.

248.—1. Cold. 2. Abd. not distd. Infln. of breasts. 4. Ut. norm. 5. 3 d. 6. 48 h. 7. R.  
Treatment, quinine.

249.—1. Feeble health. 3. Abd. distd. first few d. Sev. joints infld. 5. 4 d. 6. 19 d. 7. D. 8. 4th d. rigor, abdom. pain. Milk and loch. suppd.  
Treatment, opiates, quinine; hot fomentns. In 2nd week was found to have large abscess at back of R. forearm, also on L. shoulder and R. side of face. Sev. painful joints. Died in convulsive fit on 19th d. No P. M.

250.—3. Abd. distd. Infln. in pelvis and in R. pleura. 4. Ut. large and flaccid; loch. normal. No milk. 5. 38 h. 6. 7 d. 7. D. 8. Abd. pain on 2nd day following great effort in micturition. Local peritonitis started from L. side of abdn. (? blood forced along Fallopian tube by compression of ut. by abdom. muscles).  
Treatment, opiates, ol. terebinth.; local fomentns.; vag. injns. Later stages pointed to secondary pleurisy and implicn. of meninges. No P. M.

251.—1. Apprehension. Fatigue. 3. Abd. distd., tympanitic. 4. Ut. enlarged, sl. tender. 5. 4 d. 6. 5 d. 7. D. 8. Lab. easy, 5 hrs. Lochia scanty. 4th d. sev. rigor, fever and diarrhœa. P. 130 to 160; bilious vomiting; great thirst. Lochia suppressed. Extreme prostration. Treatment, ammonia and opium.

252.—1. Poverty, prostitution, filthy dwelling. 3. Abd. distd.; pelvic infln. 4. Ut. normal. 5. 2 d. 6. 3 d. 7. D. 8. Primipara. Child stillborn. 4th d. T. 101 M., 104 E., P. 140. Abdom. distd. and painful. Ut. and lochia normal. Delirium and great collapse preceded D. on 6th d.

Treatment, mercury and belladonna ointment; hot fomentns., poultices and leeches to abdom.; quinine and morphia internally. Good diet; stimulants.

253.—1. Had a cold previous to confinement. 2. Case of scarlat. in house 2 mos. prev. 3. Abd. distd.; pelvic infln.; peritonitis. 4. Ut. much enlarged. 5. Ab. 2 h. 6. 3 w. 7. R. 8. Lab. slow but easy. In about 2 h. sev. rigors, with much pain in abdom. and consid. distn. Several clots expelled on pres-ure over uterus. 3rd d. T. 104·2, P. 160; abd. much distd. and tympanitic; low muttering delirium. 4th d. improved. T. 102·6, P. 135. Quinine and digitalis given; linseed and mustard poultices to abd.; vaginal injns.; enema of turpentine and castor oil. 6th d. T. 99 E., much sickness; relieved by bismuth and hydrocyan. acid. 9th d. T. 101·4. 21st d. T. normal. Convalescent.

254.—1. Unmarried. Overcrowding. Attended by midwife. 3. Abd. distd.; infln. in pelvis. 4. Ut. v. painful. 5. 2nd d. 6. 6 d. 7. D. 8. Child stillborn. 2nd d. shivering, general malaise, pain in lower abd. and over uterus. 5th d. anxious, excited. T. 102·8, P. 124, dicrotic and compressible; lower abdomen painful, sl. tympanites; ut. enlarged and v. tender. Ordered opium and quinine pill; vag. syringing. 6th d. T. 102. Patient worse; delirious during night. Abdom. more distd. Hacking, painful cough, with pain in L. side, but no phys. signs in chest. 7th d. constant wandering delirium. Phys. signs in chest and abdom. unaltered. D. on 8th d.

255.—1. Twins. Large placental surface. Prolonged labour. (1st stage 1 wk.) P. p. hæmorrhage. 2. Had attended fatal case puerperal septicæmia 1 month before. 3. Abd. not distd. Consid. tenderness in pelvis. 4. Ut. normal. 5. 1st d. 6. 6 d. 7. R. 8. 1st. d. T. 104·4, P. 140. Ut. washed out twice. Quinine given. 2nd d. T. 99 M., 102 E. Ut. washed out twice. Lochia recommenced. 3rd. d. T. 100·8 M., 102 E. Treatment continued. 6th d. T. normal.

Case considered autogenetic.

256.—1. Unmarried. Depressed. 2. Puerp. case in same ward 4 mos. prev. 3. Abd. distd., painful. 4. Laceration of cervix. 5. Next d. 6. 3 d. 7 D. 8. Primip. Lab. slow but natural; placenta entire. Next d. T. 100 M., 102 E. Quinine given; ut. washed out. 3rd d. T. 103 M., 102 E. Ut. washed out twice. Sev. pain in abd. with tympanites, relieved by leeches and hot laudanum fomentns. 4th d. vomiting, P. intermitting. D. on 5th d.

257.—1. Uterine trouble after last 2 confinements. 2. Defective drainage. 3. Sl. dist. of abd. Infln. in R. side of pelvis. 4. Ut. flexed to left. 5. 4th d. 6. 6 w. 7. R. 8. First seen on 7th d., when T. 104, P. 130. From now, regular typhoid chart to end of 3rd wk.; highest T. 105. Milk and lochia suppressed. Great pain in abd., cspy. at R. side. As case progressed, great tympanites with constipation, and extreme pain on defæcation, anxiety of countenance, restlessness and high fever. Later, rigors, and great pain in R. pelvic fossa, with tenesmus and much irritability of bladder. A distinct tumour appeared to R. of ut., which increased in size daily, attended with hectic fever, profuse perspsns. and symps. of collapse. In 6th week about 1 quart of stinking brownish matter passed per rectum, followed by troublesome diarrhœa for 24 hrs. Vag. exam. showed great heat of parts, with tumour posteriorly, pressing on rectum. With disappearance of tumour came gradual convalescence.

Treatment, calomel and opium, then quinine and stimulants.



258.—1. Feeble health. 2. Attending case of erysip. at time. Placenta removed by midwife. 3. Abd. not distd. Swelling and redness of R. wrist. 4. Ut. normal. 5. 7th d. 6. 7 d. 7. D. 8. Lab. easy. 7th d. T. 103·2; violent purging and vomiting; milk suppressed. Four days later appearances seemed normal. 12th d. shivering, high fever, swelling of R. wrist. 13th d. delirium, typhoid condition, ending in D. on 14th d.

Treatment, aconite, benzoate of soda, morphia, chloral, quinine, ice.

259.—1. Many cases of cellulitis in town at time. 2. Sat up in bed on night after delivery. 3. Hands and lower part of legs and back extensively blotched with large erythematous spots. Much diffused pain over abdom. 5. Same d. 6. 7 d. 7. D. 8. Loch. v. offensive. Much abdom. pain.

Treatment, calomel and opium, quinine; vag. injns.

260.—3. Abd. distd. Infln. in pelvis. 4. Ut. large, tender. Loch. offensive. 5. 5th d. 6. — 7. R. 8. 5th d. T. 105, P. 130. Abd. distd., diarrhœa, vomiting. Loch. scanty, not offensive. Ut. not fixed. Quinine, opium and ergot given. Vag. injns. thrice daily. Turp. flannels to abd. Champagne, milk and gruel. On 8th d. discharge offensive, with shreds; ut. tender and not freely moveable. 12th d. T. 99. Lochia normal.

261.—1. Constipation. 3. Abd. not distd.; pelvic infln. 4. Ut. tender. Loch. offensive. 5. 10 d. 6. 4 d. 7. R. 8. Lab. nat. T. 104·8 on 11th d., normal on 16th d. Dyspeptic symps. for some days previously, with neglect of bowels. Tenderness in R. groin.

Treatment, calomel and jalap; hot fomentns. and poultices.

262.—1. Midwife had been feeding pigs with "swill" just before attendance. 3. Abd. distd. 4. Ut. normal. 5. 4 d. 6. 6 d. 7. R. 8. Lab. nat. Seized with rigors on 4th d. Loch. and milk suppd. T. 104, P. 140.

Treatment, mercurial purge, tinct. ferri mur.

263.—1. Primipara; protracted labour. 3. Abd. distd. 4. Ut. enlarged, painful. 5. 4th d. 6. 10 d. 7. R. 8. Began with rigors, pain in reg. of uterus. Abd. distd. Retention of urine. Highest temp. 104.

Treatment, quinine, aconite; turp. fomentns.; vag. injns.

264.—1. Albuminuria before confinement. Albumen  $\frac{1}{2}$  on previous day. 3. Rash resembling scabies all over body, esp. legs, present some while. Abd. not distd. 4. Ut. normal. 5. 3rd d. (noted). 6. — 7. R. 8. Albuminuria discovered 14 d. before labour. Confinement normal; no eclampsia. 3rd d. T. 101·8, P. 136. 4th d. T. 102·2. 5th d. T. 100·4, and from that time continued long to vary between 99° and 100°. Urine at last note from  $\frac{1}{8}$ th to  $\frac{1}{12}$ th albumen.

265.—1. Bright's disease. 3. Abd. distd.; pelvic infln. Phlebitis. 4. Ut. large, hard, tender, and v. painful. 5. 7 d. 6. 21 d. 7. D. 8. 7th d. severe rigor, followed by intense uterine pain. No apparent peritonitis. Urine sp. g. 1005, albuminous, with granular casts.

266.—1. Att. by midwife. 2. Retained and decomposing debris. 3. Abd. distd. 4. Ut. enlarged, tender. 5. 2nd d. 6. 4 d. 7. D. 8. Freq. rigors 2nd d. after del. T. 104, P. 120. Abd. v. tender and tympanitic, intense pain in lower part; frequent vomiting. Lochia suppressed. No portion of retained placenta or clot discovered.

Treatment, quinine and opium; ut. and vag. injns. Four such cases all fatal seen in neighbourhood within 3 months.



267.—1. Sev. p. p. hæmorrhage. 2. Diphth. in town at time. Both nurse and husband had sore-throat. 3. Sl. red blush over chest and trunk for a few days. Sl. congested throat. Abd. not distd. 4. Ut. norm.; loch. nat. 5. 8th d. 6. 7 d. 7. D. 8. Æt. 22; primipara. 8th d. rigors and pain behind L. shoulder. T. 105. Sodæ salicyl. given. T. fell in 2 d., then again rose to 103, and continued to fluctuate from 103 to 105. No pain. Milk ceased after 3rd d. Loch. diminishing, not offensive. 14th d. diarrhœa. D. from exhaustion on 15th d.

Treatment, salicylate, quinine, sodæ sulphocarb.; brandy.

268.—1. Highly nervous. Sev. miscarr. 3. Abd. not distd. 4. Ut. and lochia normal. 5. Next d. 6. 5 d. 7. R. 8. 2nd ch.; breech presentn.; lab. easy. 2nd d. T. 102·4. 3rd d. T. 101·6 M., 102·8 E. 4th d. T. 100·8 M., 103·4 E. 5th d. T. falling. 6th d. T. normal. Sudden rise of T. on 12th d. lasting 2 d.

Treatment, quinine, pot. brom.

269.—1. Puerperal case just opposite. 2. Ate tinned fish on morning of seizure. 3. Abd. distd. 4. Ut. norm.; loch. scanty and v. offensive. 5. 10th d. 6. 9 d. 7. D. 8. Primip. Lab. nat. 10th rigor, diarrhœa, sev. vomiting. 12th d. T. 103, P. 120, R. 60, abdom. sl. distd. and tender; loch. scanty and offensive; vomiting less. 13th d. T. 103 M., 102 E., R. 40, vom. ceased. Troublesome cough, with small crepitation over both sides of chest. Gave aconite, sp. ætheris nit., liq. ammon. acet. 14th d. T. 102·5 M., 103 E. Gave Dover's powder and quinine, mixture of cinchona ammon. carb. and æth sul. Washed out vag. 15th d. restless. T. 102 M., 103 E., P. 130, R. 30. 16th d. restless night, delirious at times. T. 102·5 M., 103 E., no pain. Catheter used. 18th d. v. delirious. Death.

Gen. treatment, stimulants; poultices; vag. injns.

270.—1. Mental depression during pregnancy. 3. Abd. distd.; pelvic infln. Bronch. catarrh. 4. Ut. enlarged, tender. Pelvic cellulitis on both sides. 5. 33 h. 6. 11 d. 7. D. 8. Lab. nat. Rigor 33 h. after del., attrib. to chill. T. 102·5. Loch. profuse 1st 24 h., never offensive. No clots. Hot vag. injns. twice daily for 1st w.; poultices to abd.; quinine and ergot first 2 d., then quinine and opium. On 5th d. unauthorized use of enema with ol. ricini, followed by violent pains in abdom., diarrhœa, great failure of strength. 8th d. stimulants, champagne, brandy; afterwards digitalis and quinine, and morphia hypodermically. D. from asthenia on 11th d.

271.—1. Small abscess near anus opened 3 d. after del. 3. Papular scarlet rash, size of hemp seed, occurred 10th w. on arms and chest. Abd. not distd. Scattered abscesses. "White leg." 4. Ut. normal. 5. About 1 w. 6.—. 7. R. 8. Primip.; good labour; sl. lacern. of perinæum. 8th d. T. 104, no rigor; no abdom. tenderness; loch. normal. 3rd w. both shoulder and both elbow joints inflamed, swollen, tender. 4th w. phlegmasia dolens L. leg. Freq. abscesses formed. No joints suppurated. Temp. varying from 100 to 105 first 6 w. Convalescence slow.

Treatment, quinine, opium, perchloride of iron. Good diet, with brandy.

272.—1. Lab. difficult. Ch. stillborn. 2. ? from case of chronic endometritis. 3. Sl. dist. of abdn. 4. Ut. sl. enlarg. 5. 3rd d. 6.—. 7. R. 8. 3rd. d. rigor. T. 103·8; great pains in abd.; loch. normal. Ordered Dover's powder, poultices to abd., vag. injns. 4th d. T. 104·8. Ut. mopped out; vag. injns. night and morning. 5th d. T. 100. 8th d. T. normal.

- 273.—3. Abd. not distd. 4. Ut. normal. 5. 18 h. 6. 15 d. 7. R. 8. 18 h. after del. rigor, severe pain over ut. 2nd d. dorsal decub., with legs drawn up; great pain over ut. and in back; great thirst. Loch. norm. Gave strong aperient; poultices to abd. Pain in back relieved. Later, calomel, James's powder; free leeching over pubic region; occas. opiates; compresses with belladonna lotion over ut. Lochia and milk abundant. Later, had consid. disch. of blood from ut., relieved by turp. and subsequently tinct. ferri.
- 274.—1. ? malaria. 3. Abd. sl. distd. 4. Ut. normal. 5. 4 d. 6. 6 d. 7. R. 8. 4th d. pain on pressure over abd. and ut. Loch. and milk abundant. Leeches applied and opiate given. 5th d. increased pain. 30 leeches applied, fomentns., poultices, and calomel given, and pain relieved. 9th d. decided symps. of intermission, with shivering, heat, and perspsns., the attack being "double quotidian," occurring at 3 a.m. and 3 p.m. Quinine in large doses with bark. 11th d. convalescent.
- 275.—1. Nervous. 3. Abd. distd. 4. Ut. flabby. Loch. suppressed 2nd day. 5. 3rd d. 6. 4 d. 7. R. 8. Septic diarrhœa on 2nd d., lasting 24 h., producing prostration and almost coma. Highest T. 104 for 3 d., reduced by quinine. Vag. injns. daily.
- 276.—2. 3 or 4 cases sore-throat in house; no membrane or rash. Scarlet. in village. 3. No rash. No sore-throat. Consid. dist. of abd.; gen. peritonitis. 4. Ut. norm. 5. 3 d. 6. 8 d. 7. D. 8. Lab. easy. 3rd d. rigor, pain over abdom. 4th d. T. 104, P. 140, abd. much distd. and tender. Loch. scanty, not offensive. Milk suppressed. Calomel and opium; turp. stupes; vag. injns. Later, quinine.
- 277.—1. Debility. Insuff. food. 2. Forceps used when head at brim; delivery slow; probably contracted pelvis. ? took chill on 3rd d. 3. Abd. not distd. 4. Ut. normal. 5. 3rd d. 6. 2 d. 7. R. 8. 3rd d. T. 103·2 M., 104·2 E., pelvic and dorsal pain. Quinine, fomentns., vag. injns. ordered. 4th d. T. 99·4 M., 100·1 E. 5th d. T. normal. Some inflammation of mammæ occurred later.
- 278.—1. Subj. to inflamm. of pleura and peritoneum. 3. Sl. distn. of abd. Sl. infln. in pelvis and in R. hypochondrium. 4. Ut. sl. enlarged, painful. 5. 3rd d. 6. 2 d. 7. R. 8. Lab. easy. 3rd d. headache, shivering, symps. of peritonitis in R. hypochondrium. Blister applied. Ev. T. 101·3, great thirst, muscular twitchings, sl. delirium. Quinine and subcut. injn. morphia given. 4th d. pain over pubes, some distn. Poultices applied, injn. morphia given, quinine and occasional brandy. 5th d. T. normal.
- 279.—2. Small pustule on thumb. 3. Abd. distd.; pelvic infln. 4. Ut. large and flabby; os patulous. Loch. nat. 5. 3rd d. 6. 4 d. 7. D. 8. Lab. easy. 3rd d. sev. pain in calves of legs. 4th d. pain also in pelvis. 5th d. a small festering pustule noticed by midwife on back of metacarpal bone of patient's thumb, with red line extending 6 in. up arm. 6th d. patient's face purple; ? pustule gone. Seen by doctor for 1st time. Opium ordered; ut. washed out. Patient much easier. 7th d. all symps. worse. No pain. Patient rapidly sank, and D. same evening.
- 280.—1. Phlegmasia dolens in a prev. confinement. 3. No rash; no sore-throat. Abd. not distd. 4. Ut. norm. 5. 3rd d. 6. 4 d. 7. R. 8. Labour nat. 3rd d. sev. headache. T. 104, P. 125, soft; sl. general tenderness, but esp. over ut. 4th d. T. 103, symps. unchanged. 5th d. T. 101. 6th d. T. norm. Treatment, salicylate. Ut. injns. on 1st d.

281.—1. Chr. endocervicitis and leucorrh. before pregnancy. Subj. to chr. gastric catarrh. 2. Attending diphth. in district same time. 15 other confinements during same month, all did well. 3. No sore-throat. Great distn. of abd.; pelvic infln. 4. Ut. enlarged, tender. 5. 3rd d. 6. Ab. 14 d. 7. R. 8. 3rd d. T. 104, had vomited freq. in night; pain in stomach; sl. tenderness over ut.; lochia sl. fœtid. Blister applied to epigast. with linseed and mustard poultices over ut.; opium and bismuth in powder; ice to suck. Vomiting ceased 4th d. T. 103 to 104 for 4 d. Tenderness first on L. then on R. of ut. A sl. pelvic tumour on R. side. Blister applied. Gradual improvement.

282.—2. Hæmorrhage from partial placenta prævia for 7 h. before del. Os. dilated with Barnes' bag, then by hand; no laceration caused. Child still-born, delivered by version. Placenta first detached 18 d. prev. Child probably died then. Epidermis separating. 3. Abd. not distd. ? pelvic infln. 4. Ut. enlarged and sl. tender. 5. 4th d. 6. 8 d. 7. R. 8. 3rd d. clots passed. 4th d. T. 101, pain in R. side of abd. (? placental attachment). Rheum. pains in wrists and fingers. Sodæ salicyl. given. 6th d. T. 104·4, P. 120, R. 26, abdom. tender, esp. in R. iliac fossa. B. O. Quinine and aconite. 7th d. T. 102·4. 9th d. convalescent.

283.—1. Intemperate during lying-in period. Insuff. food. Dirty surroundings. 2. Old laceration, extending through int. sphincter ani. 3. Sore-throat, with general exudation of clear coagd. lymph. Much distn. of abd.; pelvic infln. Vag. hot and sensitive. 4. Ut. large, v. tender; cervix thick. 5. 3rd d. 6. 7 w. 7. R. 8. Seen 3 w. after del. Then had subacute peritonitis. T. 102 to 104. Freq. bilious vomiting for 12 d. B. generally confined. Throat became sore 4 w. after del. Uvula soft palate, fauces and pharynx uniformly coated with clear lymph (not diphtheritic) for 1 w. Pot. chlor. and tinct. ferri given, with pulv. jacobini verus (of which reporter speaks most highly).

The case was considered due to intemperance during puerperal state, and this held to be a very frequent cause of puerperal pyrexia.

284.—1. Delicate. Anæmic. 3. Abd. not distd. 4. Ut. flabby; os. patulous. 5. 3 d. 6. 5 d. 7. D. 8. Lab. nat. 4th d. rigors. T. 104, P. 140; greatly depressed; sl. tenderness over ut.; loch. not offensive. Diarrhœa and vomiting preceded D. on 8th d. Treatment, ergot, iron, brandy; vag. injns.

285.—1. Nervous excitement. 3. Sl. distn. of abd. 4. Ut. normal. 5. 13 d. 6. 6 w. 7. R. 8. Lab. nat. 13th d. sudden rise of T., with pain in abd. T. remained high for 1 mo. with shifting pain, then slowly improved. Treatment, quinine, opium, chloral; stupes.

286.—1. Sl. adhesion of membr. in prev. labours. Mental anxiety. 3. Sl. dist. of abdn.; pelvic infln. 4. Ut. large; later, fixed, with swelling around. 5. 4th d. 6. 21 d. 7. R. 8. 4th d. sev. rigor. 5th d. T. 102 M., 103 E. 6th d. T. 104. 7th d. T. normal. 10th d. T. 104·5, and remained high till end of 3rd w. Rigors on several occasions. Tenderness on pressure above pubes and on vag. exam. Treatment, opiates, salines; poultices; ut. and vag. injns.

287.—1. Anæmia. Debility from 7 prolonged lactations and as many sev. p. p. floodings. Apprehension. 3. V. sl. dist. of abdn. 4. Ut. sl. large, sl. pain on pressure. Loch. and milk normal. 5. Next d. 6. 4 w. 7. R. 8. Symptoms of flooding occurred 7 h. after del., but were checked by sp. terebinth. T. seldom below 101 till late in 4th w. after del. Treatment, quinine, iron (2nd wk.). No stimulants.



288.—2. Laceration of vag. and perinæum. 3. Abd. not distd.; sl. pelvic infn. 4. Ut. tender. 5. 10 d. 6. 12 d. 7. R. 8. Much rambling first 4 d. T. from 102 to 105.  
Treatment, quinine.

289.—3. Rash over whole body, exc. hands. No sore-throat. 4. Ut. at first normal, later relaxed. Loch. nearly ceased. 5. Ab. 3rd d. 6. 2 d. 7. D. 8. Primip. Good labour. Sl. cold before confinement. Did well for 2 d. 3rd d. T. 100, great thirst. 4th d. general rash, and within 24 hours death, the rash first disappearing.

290.—3. Miliaria over whole body. Abd. not distd. 4. Ut. relaxed. Loch. arrested before app. of rash. 5. 2 or 3 d. 6. Ab. 24 h. 7. D. 8. Labour tedious. About 3rd d. ate largely of salad. Soon after, vomited severely. Loch. ceased, and in a few hours a bright miliarious rash appeared. Rash entirely gone before D. of patient on following day.  
Treatment, belladonna, ammon. acet.

291.—3. Florid miliary rash over whole body. Abd. sl. distd. 4. Ut. firm. 5. 4th d. 6. 4 or 5 d. 7. R. 8. Primip. 39; labour protracted. 4th d. loch. suppressed. A profuse sudaminous rash appeared a few hours later and lasted a few days. Much insomnia.  
Belladonna, chloral and bromide given.

292.—3. Abd. distd.; pelvic infn. Phlebitis R. femoral vein. 4. Ut. enlarged, tender. 5. 3 d. 6. 4 d. 7. R. 8. Fever, with occas. rigors. Abdom. tenderness and pain, with vomiting and loch. suppressed. In 10 d. phlebitis followed by abscess.

Treatment, quinine, ergot and digitalis, iron being added when rigors contd. and abscess set in. Locally, uterine injns. for first few days, then carbolyzed pledgets applied to cervix.

293.—1. Scrofulous. 3. Abd. distd.; pelvic infn. 4. Ut. tender and swollen. Cellular tissue round cervix much infiltrated. 5. 2 d. 6. 2 m. 7. R. 8. T. 104, rigors, quick pulse, profuse perspsns., vomiting and delirium. Loch. suppd. some days. Extreme uterine tenderness. All symps. pointing to acute metritis and cellulitis, ending in 2 mos. in a gluteal abscess, which healed in 6 w.

Treatment, quinine, digitalis, ergot and iron internally. Locally, uterine injns. twice daily. These were more than once after the 4th d. followed by rigors and delirium (? fresh surface for absorption opened up in uterus), and a vaginal plug of lint soaked in carbolic acid was therefore used.

294.—1. Illegitimacy causing nervous excitement. 3. Abd. distd.; pelvic infn. 4. Ut. swollen and tender. 5. 2 d. 6. 1 m. 7. R. 8. High fever, with vomiting, extreme uterine tenderness and pain, and lochia suppressed.

Ut. injns. twice daily first few days, with quinine, digitalis, iron and ergot internally. Use of injns. in this and in 2 other cases followed by rigors after first few days, though v. carefully administered.

295.—2. One of med. attendants was attending cases of febricula. 3. Sl. rash on chest. Sl. sore-throat. Abd. not distd. 5. At once. 6. 8 d. 7. R. Desquamation. 8. 2nd and 3rd d. T. 104, P. 140. Both falling gradually to normal by 8th d.

296.—1. Delicate. 3. Abd. distd. Pelvic infn. 4. Ut. large, soft flabby. 5. 5 d. 6. 62 d. 7. R. 8. No complication. Syringing till 22nd d.

297.—1. Delicate. 3. Abd. distd. Pelvic infn. 4. Ut. large, soft flabby. 5. 4 d. 6. 12 d. 7. R. 8. Treatment, quinine, salicylates; ut. injns.



298.—2. No injection. 3. Abd. distd. Pelvic infln. 4. Ut. large, soft, flabby. 5. 4 d. 6. 20 d. 7. R. 8. Treatment, quinine, salicylates; ut. syringing.

299.—2. Supposed to have caught cold. 3. Abd. distd. Pelvic infln. 4. Ut. large, soft, flabby. 5. 4 d. 6. 20 d. 7. R. 8. Began with rigors and fever.

Quinine and salicylate given, with free ut. syringing. Recovery complete.

300.—1. Et. 20, very delicate. Labour 2 w. prem., during attack of pleurisy. 3. Abd. distd.; infln. in pelvis and elsewhere. Ut. —. 5. Concurrent. 6. 7 d. 7. D. 8. Complication with pre-existing disease veiled special symptoms. No infection possible.

301.—1. Depression. 2. Chr. ulcer. of uterus. 3. Abd. dist. Peritonitis. 4. Ut. large, soft. 5. 3rd d. 6. 7 d. 7. D. 8. Lab. easy. 3rd d. T. 103; sickness and diarrhœa; abd. much distd. and v. tender. Loch. suppressed, no clots. T. remained high till D.

Treatment, ergot, opium, quinine, dil. sulphuric acid, sodæ hyposulph.; turp. stupes; ut. injns.

302.—1. Many prev. confinements. 2. Scarlet. prev. in district during previous quarter. 3. R. labium red and swollen on 4th d. Abd. distd. 4th and 5th d. Some swelling back of L. leg on 5th d. 4. Ut. enlarged, sl. tender. 5. 30 h. 6. 4 d. 7. D. 8. Had a chill 24 h. after del. T. in 30 hrs. 101. 2nd d. T. 99·4 M., 103 E. Quin. and opium; turp. stupes, poultices; vag. injns. 3rd d. T. 101·4 M., 103 E. Opium and frequent doses quinine; vag. injn. 4th d. T. 103 M., 104·8 E. Quin. contind.; ut. injn.; ice-cap applied. 5th d. noon T. 105·4. Ur. contained much albumen. Death in evening.

303.—1. Delicate. 2. Careless nursing. 3. Abd. not distd. Limited infln. about nt. 4. Ut. normal. 5. 3rd d. 6. 22 d. 7. R. 8. 3rd d. T. 102·4 M., 103·4 E. On 7th d. T. normal, rising again on 10th d. when solid food given by nurse. 12th to 15th d. T. 100 to 103·4, though frequent doses quinine and aconite given and vag. injns. used. 15th to 22nd d. T. varying up to 104·5. B. O. thrice daily. No spots; no tenderness. Sl. feeling of resistance per vag. on one side of ut., with some tenderness here on pressure. Ut. not fixed. T. normal on 23rd d. Much supra-orbital neuralgia during conv.

304.—3. Abd. sl. distd. 4. Ut. ? 5. 10th d. 6. 2 d. 7. R. 8. Primip.; lab. tedious. 10th d. rigor; sev. abdom. pain. T. 103·4, P. 140. Aconite, hot fomentns. 11th d. T. normal, no pain.

305.—3. Some distn. of abd. Infln. extending above brim of pelvis and in L. iliac region. 4. Ut. ? 5. 8th d. 6. 10 d. 7. R. 8. 8th d. shivering, pain in lower abdn. T. 103·8, loch. offensive. 11th d. T. 104·6. 13th d. T. 105·2, then fell gradually. Tumefaction later in L. iliac fossa and roof of vag.

Treatment, aconite, quinine; vag. injns.

306.—3. Abd. distd. 4. Ut. normal. 5. 20 d. 6. 4 d. 7. D. 8. Primip.; lab. easy. Went out to fields 14 d. after del. 20th d. vomited; complained of numbness in arms. 22nd d. T. 106, P. 140 to 160; hdchc.; occas. vomiting; no abdom. pain.; loch. natl. D. on 23rd d.

Treatment, calomel, opium, quinine.

307.—1. Delicate. 2. Sl. lacern. of perinæum. 3. Abd. not distd. 4. Ut. normal. 5. 48 h. 6. 4 w. 7. R. 8. Primip.; labour natural. Fever 48 h. after del. No pain. Loch. scanty; no milk. Highest T. 103 (3rd d.) P. 130, gradually subsiding. Erythematous spots over finger joints 3rd week.

Treatment, salines, quinine, ferri perchlor.; ut. injns.

308.—2. Uncleanliness after del. No nurse. Attending scarlat. same time. 3. Dull red papular rash on neck, shoulders, arms. No sore-throat. Abd. sl. distd. 4. Ut. sl. tender. 5. 4th d. 6. 2 d. 7. R. 8. Primip. Sl. lacern. of perineum. 3rd d. breasts full. 4th d. rash appeared. Gave salines, vin. colchici, bromide. 5th d. T. 105. Loch. present. Mag. sulph. given. 2 d. later T. normal; no rash; could suckle child.

309.—3. Abd. distd. Infln. in pelvis and general in abd. 4. Ut. large, subinvolved. Loch. not offensive. 5. 3rd d. 6. 2 d. 7. D. 8. Lab. normal. 3rd d. diarrhœa, continuing after castor oil, with sickness and prostration. 4th d. restlessness, delirium, vomiting; abd. distd.; pain about navel, severe before D. Died collapsed same evening. No P. M.

310.—3. Abd. sl. distd. Abscesses in L. elbow, wrist and ankle. 4. Ut. soft, boggy. 5. 24 h. 6. 74 d. 7. D. 8. Primip. Bruising of vag. fr. tedious labour. Acute ut. pain in 24 h. 2nd d. rigor. T. 104. On 17th d. T. 106. Acute pain in L. hip from about 20th d. From 30th d. onwards T. varying from 99 M. to 104 E. Sev. small abscesses formed.

Gen. treatment, quinine, Warburgh's tincture, perchloride of iron, ammonia and bark; ut. and vag. injns.; morphia; cold sponging, wet packing; stimulants.

311.—1. Breech case. 3. Abd. distd.; infln. in pelvis; gen. peritonitis. 5. 2 d. 6. 4 d. 7. D. 8. Began with pain, tenderness, tumidity in hypogastrium. Rapidly became worse. High fever, vomg., delirium, abd. much distd. D. on 4th d.

Treatment, aconite and opium; morphia suppos.; poultices; vag. injns.; ice. Baby d. of septicæmia when 11 d. old; sev. joints inflamed. Suckled by M. on day of her death.

312.—3. Abd. sl. distd. and v. tender. 4. Ut. large. 5. 10 d. 6. 2 d. 7. R. 8. Did v. well till 10th d., when severe rigor and pain over R. ovary. T. 104 M., 105·6 E.

Gave salicine; mopped out vag. and ut. with glyc. ac. carbol.; poultices to abd. Improved rapidly.

313.—3. Sl. distn. of abd.; pelvic infln. 4. Ut. tender; loch. scanty. 5. 36 h. 6. 8 d. 7. R. 8. 3rd d. morn. T. 104·5, P. 130; even. T. 102, P. 115. Great pain in lower abd. Linsced poultices, opium. 7th d. T. normal. 8th d. T. 101·5. 10th d. T. normal. There was violent delirium for a few days.

314.—3. Abd. not distd. R. mamma infld. on 2nd day. 4. Ut. norm. 5. 11th d. 6. 3 d. 7. R. 8. Extreme vomiting and fever. R. breast inflamed 2nd d.

Treatment, leeches, hot fomentns., warm oil, aperients, imperial drink.

315.—1. Apprehension as to result. 2. Had seen case of peritonitis same day. 3. Abd. distd. Local infln. L. side of pelvis, becoming general peritonitis. 5. 30 h. 6. 9 d. 7. D. 8. Primip.; natural labour. After 30 h. got up and at once complained of shivering and pain in L. side. Ut. well contracted, but great tenderness on L. side. T. 102, P. 130. Opium and ammonia given; hot applics. to side. Became gradually worse. Abdom. greatly distended and tender; loch. offensive. Milk never appeared. Ut. and vag. injns. used. Fever increasing, wet pack used twice, with reduction of fever for 6 hrs. P. gradually became weaker. D. insensible on 9th d.

Two other cases (not fatal) directly infected from this case.

316.—1. Ill-health. Abraded os ut. and congested cervix before pregnancy. 3. Papular eruptn. on neck and hands, like lichen. Abd. n. distd. Tenderness L. side of pelvis 1 d. 4. Ut. and loch. normal. 5. 56 hrs. 6. 11 d. 7. D. 8. 3rd d. sev. rigor, T. 103, P. 130. 5th d. T. 99. 6th d. T. 103, P. 100. 8th d. T. and P. normal, feeling well. 9th d. T. 104, P. 116. 12th d. morn. T. 105, delirium, diarrhoea. D. on 12th d.

Treatment, quinine, sodæ salicyl.

317.—1. Prev. ill-health. 3. Painful ulcerated throat. Sl. distn. of abd. 4. Ut. enlarged, tender, fixcd. 5. 2nd d. 6. About 10 d. 7. R. 8. Sore-throat before labour. 2nd d. T. 103-104. Abdom. tender. Loch. suppressed. Pain long persistent. Conv. slow.

Treatment, diaphoretics, opium, pot. brom.

318.—3. Labial herpes on 4th d. of fever. Abd. distd. 4. Ut. v. tender. 5. 5th d. 6. 8 d. 7. R. 8. Labour easy. 5th d. eveng. severe rigors, followed by ut. pain and tenderness; lochia and milk suppressed. 6th d. copious sweating, followed by fall of T. and P. with reapp. of milk and loch. 7th d. evg. rigor, fever; loch. again suppressed. 8th d. T. 105·8, delirium, perspn. T. falling to normal. 9th d. evg. rigor, with great ut. tenderness; copious herpes on lips and nose. 10th d. diarrhoea, profuse sweatings. 12th d. slept well. T. falling. 14th. d. T. subnormal; resumed nursing child.

Treatment, large doses quinine; stupes and poultices. Milk diet.

319.—3. Abd. not distd. Leg inflamed. 4. Ut. normal. 5. 4 d. 6. 4 w. 7. R. 8. Ch. stillborn. 4th d. symps. of phlegmasia dolens in L. leg with sl. fever.

Treatment, quinine and digitalis. Local applicns. of hot poppy fomentns., belladonna, and finally blisters. Good diet; stimulants.

320.—1. First confinement. Apprehension. 2. ? great constipation 3. Abd. distd., painful, tender. Pleuropneumonia. 4. Ut. large until disch. of clots 12 h. after labour. 5. 6 d. 6. 10 d. 7. D. 8th d. rigor, vomiting. T. 102, P. 120, pain and tenderness over lower abdn. Great improvement in next few days. 10th d. increase of fever, phys. signs of pleuropneumonia on R. side. Much worse during next four days. 15th d. rapid improvement; less pain; no vomiting; tongue cleaning. T. 100, P. full and slow.

Calomel given in eveng. by another medical attendant. Violent diarrhoea followed. Patient became comatose and died on 16th day.

321.—Unmarried. 3. Abd. dist.; pelvic infln. 4. Ut. swollen, tender. 5. Next day. 6. 1 mo. 7. R. 8. Primip.; labour prolonged; forceps used. Next day, rigors, followed by pain and tenderness over ut., quick pulse; hot skin; loch. suppressed. Gave calmel and opium, salines; ut. and vag. injns. 6th d. much less pain.

Bark and ammonia given, with morphia at night. Convalescent 4th week.

322.—2. Had nursed 2 children with fatal diphth. and purpuric eruption 3 mos. before delivery. 3. Crimson erythematous rash on abd. 4th day purpuric spots on trunk and extremities. 6th day no sore-throat. Abd. distd.; pelvic infln.; general peritonitis. Pneumonia. Ut. soft, not large. Loch. normal. 5. 14th d. 6. 8 d. 7. D. 8. Labour easy. Was up 10th day. 13th d. pain over L. ovary, not elsewhere. No milk. Loch. normal. 14th d. rigor, fever. P. 120, tongue dry; hdche.; vomiting. Next 3 days increasing ovarian and uterine pain; general peritonitis, persistent vomiting, and increased exhaustion. On 17th d. crimson rash appeared. 18th and 19th days all symps. aggravated. Advent of double pneumonia. 20th d. delirium, purpuric eruption and death.

Treatment, opium, with diaphoretic salines; morphia injns.; turp. stupes to abd.; vag. injns. Frequent fluid nourishment, with free brandy.



323.—1. Intemperance. 3. No distn. of abdn. 4. Ut. swollen, tender and painful. 5. 2 d. 6. 10 d. 7. R. 8. Primip. Much excited and sleepless. 3rd d. fever, delirium. Loch. and milk partially suppressed. V. slight signs of local infln.  
Treatment, bromide and chloral.

324.—1. Mental worry. 3. Abd. sl. distd.; pelvic infln. Mammary abscess. 4. Ut. large, tender, painful. 5. 10 d. 6. Many weeks. 7. R. 8. Æt. 18; unmarried. Up on 9th d., but gave evidence at once of metritis.  
Opium and quinine given; uterine and vag. injns.; stimulants.

325.—1. Intemperance. 3. Abd. distd.; pelvic infln. 4. Ut. large, tender, painful. 5. 4 d. 6. 10 d. 7. R. 8. Forceps used for inertia. 3rd d. retention of urine. Catheterised daily. 5th d. T. 105, ut. swollen, tender and painful; loch. suppressed. T. varied much. Rapid recovery.  
Treatment, bromide, chloral, morphia; uterine and vag. injns. No stimulants.

326.—1. Insuff. food. 3. Abd. distd.; pelvic infln. 4. Ut. large, tender, painful. 5. ? 6. 1 mo. 7. D. 8. Ut. alone showed signs of infln. Death from asthenia.  
Treatment, stimulants, good nourishment.

327.—2. Tedious labour; much manual interference; forceps used. 3. Abd. not distd. Hypogastric tenderness. 4. Ut. painful, tender. 5. 3rd d. 6. 7 d. 7. R. 8. 3rd d. T. 104. 8th d. T. normal.  
Quinine, opium; ut. and vag. injns.

328.—3. Abd. not distd. Great tenderness over hypogastm. and of uterus on vag. exam. 5. 4 d. 6. 8 d. 7. R. 8. Primip.; labour 17 hrs., normal. 4th d. fever and abdom. tenderness. For next 6 d. T. 102 to 104, increased abdom. tenderness. Loch. from first scanty, not offensive. Milk freq. 10th d. uterine injn. 11th d. T. fell 4 deg. and was normal next day.  
Treatment, small doses of quinine with digitalis. Vag. injns. daily.

329.—1. Mental depression. Illegit. child. 3. Mod. distn. of abdn., pelvic infln. Acute bronch. 4. Ut. large and v. tender. 5. 5 d. 6. 16 d. 7. R. 8. Lab. prolonged; forceps used. 5th d. rigor. T. 102·5, severe hdche.; abdoml pain. 8th d. loch. ceased. Sl. purulent disch. on 9th d. Severe bronchitis on 12th d.  
Treatment, quinine and opium; hot fomentns.; uterine injns.

330.—1. Highly nervous. Great dread of confinement. 4. Ut. norm. Loch. norm. 5. 27 h. 6. 5 d. 7. R. 8. 1st ch. Labour v. prolonged. 2nd d. morn. restless. T. 94, P. 94. Bromide and chloral given. Eveng. no sleep; restless. T. 103·4, P. 120, tongue coated; abdom. tender, loch. natl. Repeated bromide and chloral. Ol. ric. in morning. 3rd d. m. Has slept 2 hrs. T. 101, P. 108. Feels better. Haust ammon. acetatis. In eveng., no sleep through day, T. 105·6, P. 140, skin dry; muttering delirium. Small freq. doses aconite and digitalis. 4th d. profuse sweat last evng. Feeling weak. T. 99·5, P. 96. Hence slow but good recovery.

331.—1. Apprehension. 2. No infection. Forceps used. Have had no other fatal case before or since. Drainage bad. 3. Abd. dist.; pelvic infln. Hypostat. pneumonia later of bases both lungs. 4. Passages hot and irritable. 5. 3 d. 6. 16 d. 7. D. 8. 3rd d. high fever, rapid pulse; abdom. tender and swollen. Later, diarrhoea and hypostat. pneumonia both bases. Gradual supervention of typhoid state. D. on 19th d.  
Treatment, small doses quinine, with morphia; vag. injns. Later, opium, ammonia and belladonna. Stimulants. Nourishing diet and drinks.



332.—1. ? sl. lacern. of cervix. 3. Mod. distn. of abdom.; tenderness in lower part. 4. Ut. sl. enlarged. 5. 3rd d. 6. 3 d. 7. D. 8. Primip. 22; labour easy. 3rd d. fever, vomiting. Bowels loose. In eveng. v. delirious; loch. ceased. 4th d. T. 103, constant vomiting and purging; abdom. dist. and tender. 5th d. coffee grounds vomit.; rapid collapse. All treatment ineffectual. No lues venerea. Ch. d. 14th d. with suppurating phalangeal joints one hand, and swelling of one ankle. Had also growth like mucous tubercle at anal margin.

333.—1. Mental depression. Ch. illegit. 3. Skin sallow. Abd. dist.; pelvic infln. at outset. 4. Ut. lax. 5. 1 w. 6. 3 w. 7. D. 8. Began with hdche. and occasl. shiver. Uterine pain. Loch. offens. Treatment by acid sulphuros. and bark; ammonia and bark; vag. injns.; stimulants.

334.—2. Slight laceration of perineum. 3. Abd. much dist. Left iliac peritonitis. 4. Ut. large and tender. 5. 3rd d. 6. Ab. 18 d. 7. R. 8. Primip.; labour easy; after-pains severe for 24 h. Eveng. 3rd d. rigors. Milk soon suppd. Loch. scanty and offensive. Abd. distd. and tender, esp. in L. iliac region. T. from 101 to 105. Treatment by quinine.

335.—1. Puerperal fever in prev. pregnancy. 3. Abd. distd. and tender. 4. Ut. painful; loch. suppd. 5. Next day. 6. 7 d. 7. D. 8. Forceps used for inertia. Ch. stillborn. 2nd d. rigor, fever, vomiting; abd. much dist. and tender; lochia suppd. 5th d. diarrhoea, muttering delirium. 7th d. D.

Treatment, salines, opium; vag. injns.; turpentine stupes. Careful diet.

336.—1. ? old malarial poisoning. No illness 3 y. 4. Ut. nat. 5. 4 . 6. 10 d. 7. R. 8. Continued high temperature (104), with insomnia and occas. rigors, readily yielding to quinine. Similar condition noted in a later confinement.

337.—2. Bad nursing. ? chill. 4. Ut. normal. 5. 7 d. 6. 3 d. 7. R. 8. Evening 7th d. T. 104, intense headache; back-ache. 10th d. T. fell, with relief of symps.

Treatment, quinine, morphia; vag. injns.

338.—No infection. 3. Abd. distd.; pelvic infln. 4. Ut. large, tender 5. 40 h. 6. 2 wks. 7. R.

Treatment, opium; fomentns.

339.—3. Abd. distd. 4. Ut. normal. 5. 4 d. 6. 30 d. 7. R.

Treatment, mercurial purge, large doses of tr. ferri perchlor.

340.—1. Ruptured perineum. 3. Herpes on lips. Abd. distd. Breasts and vag. infld. 4. Ut. normal. 5. 2nd d. 6. 10 d. 7. R. 8. Lab. natural. Ruptured perineum. 3rd d. slight fever. 5th d. T. 102, P. 120. 6th d. fever still high, rigors. 7th d. T. 100, P. 120. 8th d. enema omitted. 9th d. T. 103, P. 130. 12th d. T. and P. norm.

Treatment, mercury, antimony, salicylic acid, quinine and opium; ut. and vag. injns.

341.—3. Abd. dist. Perimetritis. Peritonitis. 4. Ut. large, soft, tender; os patulous. 5. 3rd d. 6. 7—9 d. 7. R. 8. Labour normal. 3rd d. morn. T. 102, P. 120, small, thready; vomiting; diarrhoea; severe pain in back and abdom.; tymp.; lochia and milk suppd. 3 h. later severe rigor, T. 106.2. Quinine sulph. grs. 20; frequent iced packs to head, chest and thighs. Quinine and opium every 4 h. Cold packs when necessary. T. 101 to 102 for 9 d.

Ut. and vag. injns. used throughout.

342.—1. Depression. 5. 24 h. 6. 1. 7. R. 8. Labour normal. 2nd d. T. 101. No clot. Loch. normal. Nymphæ much swollen. T. long, varied from 100 to 104 without apparent cause, once reaching 105·6. Slight phlebitis left fem. v. 6th w.

Treatment, salines, quinine, sodæ salicyl., digitalis.

343.—1. Men'al anxiety. Mother had nursed 3 ch. with scarlat. 6 mos. prev. 3. No rash. No sore-throat. Tympanites on 12th d. 4. Ut. normal. 5. 6th d. 6. 9 d. 7. D. 8. Version necessary. Child stillborn. 6th d. violent diarrhœa, with symps. of collapse. T. in evg. 102·5. Patient rallied after opium. No further purging. T. remained above 100. D. on 15th d.

Treatment, opium; antiseptic uterine injns.

344.—1. Albuminuria. Eclampsia. 2. Lochia partially suppd. 2 d. after delivery. 3. Abd. dist.; slight peritonitis. 4. Ut. normal. 5. 2 d. 6. 2 d. 7. R. 8. Several severe convulsions before labour. Under chloroform labour induced and forceps used. Unconscious 2nd and 3rd and part of 4th d. T. normal. During this time fed per rectum. Chloral and pot. brom. given. 4th d. T. 102·5, abd. slightly dist.; seemed in pain; ut. enlarged. Turpentine stupes to abd. 5th d. T. 101·5, rather more disch. Same treatment. 6th d. T. 99·5. Conscious. 7th d. T. normal. Chloral and bromide at night. No fit after 5th d.

345.—2. Made P. M. exam. on case of cancer of liver with peritonitis 1 w. previously. 3. Abd. dist.; local infln. of pelvis; peritonitis. 4. Ut. subinvolved. 5. 22 h. 6. 7 d. 7. D. 8. Primipara; labour easy; forceps used. Same evg. T. 103·2, pain and tenderness over abd. Gave opium and vag. injns. 3rd d. morn. less pain and tenderness. Tinct. ferri perchlor. and opium. 4th d. T. 103·4, great pain. Nit. mur. dil. given. 5th d. T. 103·5, P. weak; rambling. 6th d. T. 102·5. Pulse scarcely felt. Stimulants increased. 7th d. T. 100·3, tongue dry, with brown fur. Still rambles; picks bedclothes; purged often during day. In evg. slight convulsions; death.

P. M. exam. showed signs of recent general peritonitis. Pus in pelvic cavity.

346.—1. Nervous depression. Fear of fatal termination. 3. Abd. not dist. 4. Ut. normal; slight laceration of cervix. 5. Same day. 6. 4 d. 7. D. 8. Primipara. 30. Married 5 yrs. Labour slow; forceps used, slight laceration of perineum; mod. p. p. hæmorrhage. Same evg. T. 99·6. P. 108, patient exhausted; vomited. 2nd d. complaining of want of breath: thirst. 3rd d. sickness; slight jaundice. T. 99·9, P. 128. In night semi-comatose. 4th d. T. 98, P. 120. Continued unconscious through day; everything passed under her; abd. dist.; loch. somewhat offensive. D. on 5th d.

Treatment, vag. injns., good diet, stimulants.

347.—1. Subject to malarial fever. 2. Memb. adherent; had to be removed. 3. Abd. not dist. 4. Ut. normal. 5. 4th d. 6. 6 d. 7. R. 8. Labour rapid. Membræ adherent; carefully removed. 4th d. T. 103·4, P. 120, and so remained (circa) till 8th d. No rigors. Tongue white and tremulous. 9th d. T. normal.

Treatment, quinine in 1 gr. doses; local disinfectants.

348.—1. Primip. Twins. 3. Abd. dist. Infl. in pelvis and in left calf (abscess). 5. 3rd d. 6. 17 d. 7. R. 8. Easy labour. 4th d. T. 103·5, P. 125. 5th d. T. 105·5, P. 120, R. 28. 6th d. T. 101, P. 120. 7th d. T. 100, P. 95. From this till 16th d. T. about 103. 9th d. tympanites; slight delirium. 11th d. purulent vag. discharge; little fœtor. 13th d. still tympan.; commencing bedsores. 17th d. morn. T. 105, P. 140. Both normal in evg. B. O. freely. 22nd d. T. and P. normal, and so continued. Sleeps well. Abscess opened in leg. 29th d. no vag. discharge; sits up. 35th d. gets up. Convalescent.

Treatment, opium, quinine, poultices; vag. injns.

349.—1. Hard work. Mental distress. 3. Abd. much distd. towards close. 5. 7th d. 6. 5 d. 7. D. 8. Multipara. Labour easy. D. fr. effusion on brain, with gen. paralysis. T. usually 105, rose above 108° shortly before death. No pain throughout illness. Expression maniacal; rambling; great heat of head. Nil in pelvis, abdomen, or chest.

350.—1. Post partum hæmorrhage; ferri perchlor. injected. 3. General sudaminous rash. Abd. not distd. 4. Ut. sl. tender. 5. 2nd d. 6. 12 d. 7. R. 8. Insomnia. Severe rigors 2nd and 3rd d. T. 104°–105°. Sl. delirium.

Ut. washed out regularly with 1 in 60 solution of carbolic acid and warm water. Salicylate of iron internally. Beef-tea, gruel, port wine.

351.—1. Forceps used in sev. previous labours. 2. Doctor attending severe enteric case at time. 3. No rash; no sore-throat. Abd. distended. Well marked metritis. Pneumonia. 4. Cervix torn. 5. Next day. 6. 10 d. 7. D. 8. Outlet narrow; forceps used; perinæum entire. High fever. T. av. 103·5. Urine drawn off twice daily. 1 wk. after delivery sloughs and much pus discharged from vagina. Sank gradually with signs of septicæmic poisoning.

Treatment, calomel and opium; turpentine stupes, poppy fomentations.

352.—1. ? chronic ulcer of leg. Room small. 3. Abd. distended. Considerable effusion on right side of ut. Pus in left knee joint. 4. Uterus large and v. tender. Lochia scanty and foul. 6. Protracted by state of knee; primary fever about 10 d. 7. R. 8. Labour normal. Restless same night. 2nd d. T. 101°. 4th d. 1st flushed; pain in abd.; effusion felt in R. side of pelvis (per vag.). Ut. tender; loch. scanty, offensive. T. 103°. 7th d. furious delirium; infln. in L. knee, much pain. Pelvic symps. improved. Abscess formed in knee joint; this opened and drained. Recov. with stiff joint in 4 mos.

Treatment, sod. salicyl.; vag. injns.

353.—1. Mental depression. 3. Abd. normal. 4. Ut. tender. 5. 4th d. 6. 3 d. 7. D. 8. Child stillborn. Placenta normal. 4th d. Pain and tenderness over whole abdom.; anorexia, thirst, constipation; loch. scanty and foul; tongue furred, moist. T. 101, P. 110. 5th d. tongue drier. T. 101·5, P. 120, easily compressed; loch. suppressed; severe abdom. pain. Turpentine stupes. 6th d. tongue brown. T. 101·5, P. intermitting. Still severe pain. Face drawn and pinched. 7th d. tongue v. brown. T. 100, P. quick, intermitting. Severe paroxysmal pain. Collapse. Death.

Treatment, calomel, opium, chloral; poultices; local injns.

354.—1. Previous ill-health. 2. Insan. conditions. 3. Sl. distn. of abd. Metritis. Sev. pain over liver on 7th d. 4. Ut. large, v. tender. 5. 3 d. 6. 8 d. 7. D. 8. 3rd and 4th d. T. 104·8. 5th d. T. 103·2, then fell gradually to normal on 8th d. Diarrhœa. D. on 11th d.

Treatment, salicylate, quinine, opium; vag. injns.

The Committee beg to acknowledge reports of cases of fever in the puerperal state received from the following gentlemen. They were received too late to be included in the subject matter of the present preliminary report:—

George T. McKeogh, Ontario; T. Corbett, M.R.C.S., Kingston-on-Thames; G. Wyndham Crowe, M.D., Worcester (2); J. Campbell, M.B., Elcheater (2); Edwin Child, M.R.C.S., New Malden (2); Forbes Dick, M.D., Surgeon-Major A.M.D., Burmah (5); Reginald Bayley, L.R.C.P., Kingston; J. W. Miller, M.D., Dundee; A. D. Leith Napier, M.D., Dunbar, N.B. (9); J. J. Stack, Knighton; James Donald, Kingston-on-Thames; J. Hurd Wood, M.D., Letherhead; George G. Whitwell, M.D., Shrewsbury; G. H. Charlesworth, L.S.A., Waudsworth; Charles P. Hooker M.D., Coltishall.



## Organisation for the Collective Investigation of Disease.

### COLLECTIVE INVESTIGATION COMMITTEE OF THE ASSOCIATION.

Professor Humphry, F.R.S. (Chairman); C. G. Wheelhouse, F.R.C.S. (President of Council); W. F. Wade, M.D. (Treasurer); R. L. Bowles, M.D.; A. Carpenter, M.D.; B. Foster, M.D.; C. Macnamara, F.R.C.S.; A. Ransome, M.D.; F. A. Mahomed, M.B.; W. P. Herringham, M.B. (Secretary).

### GENERAL COMMITTEE.

Thomas Aitken, M.D.; Thomas Barlow, M.D.; H. T. Butlin, F.R.C.S.; T. Lauder Brunton, M.D.; W. E. Buck, M.D.; J. Cavafy, M.D.; W. B. Cheadle, M.D.; Sidney Coupland, M.D.; J. Ward Cousins, M.D.; A. Davidson, M.D.; N. Davies-Colley, F.R.C.S.; Dyce Duckworth, M.D.; G. Eastes, M.B.; F. Galton, F.R.S.; F. J. Goodhart, M.D.; T. H. Green, M.D.; W. C. Grigg, M.D.; J. W. Howard, F.R.C.S.; J. Hutchinson, F.R.C.S.; G. B. Longstaff, M.B.; Sir W. MacCormac, Bart., F.R.C.S.; Stephen Mackenzie, M.D.; Withers Moore, M.D.; Shirley F. Murphy, Esq.; H. Page, F.R.C.S.; C. Palmer, Esq.; A. Parsons, M.D.; Rees Phillips, M.D.; S. J. Sharkey, M.B.; Octavius Sturges, M.D.; Fred. Taylor, M.D.; C. Turner, M.D.; W. J. Tyson, M.D.; J. Burney Yeo, M.D.; and the local Hon. Secs., who are *ex officio* members of this Committee.

### LIST OF LOCAL COMMITTEES IN THE BRANCHES,

*With Returns sent, up to June 30, 1884.*

#### Aberdeen, Banff, and Kincardine Branch.

*Hon. Sec.*—J. Mackenzie Booth, M.D., Union Street, Aberdeen.

\* *Returns.*—I., 9; II., 3. Total, 12.

Total returns from the Branch, 12.

#### Bath and Bristol Branch.

BATH DISTRICT.—*Hon. Sec.*—R. J. H. Scott, Esq., 13, Bladud Buildings, Bath.  
*Returns.*—I., 1; VII., 1. Total 2.

BRISTOL DISTRICT.—*Hon. Sec.*—E. Markham Skerritt, M.D., Richmond Hill, Clifton.

*Returns.*—I., 8; III., 2; IV., 2; VII., 2; X., 1. Total, 15.

Total returns from the Branch, 17.

\* I., Pneumonia; II., Chorea; III., Rheumatism; IV., Diphtheria (Clinical); IVa., Diphtheria (Sanitary); V., Syphilis (Acquired); Va., Syphilis (Inherited); VI., Acute Gout; VII., Puerperal Pyrexia; VIII., Paroxysmal Hæmoglobiuria; X., Habits of Aged Persons; XI., Albuminuria in the apparently healthy.



**Birmingham and Midland Counties Branch.**

*Chairman.*—B. Foster, M.D.

*Hon. Sec.*—Robert Saundby, M.D., 25, Newhall Street, Birmingham.

*Returns.*—I., 7; II., 1; IV., 1; VII., 3; X., 1. Total, 13.

Total returns from the Branch, 13.

**Border Counties Branch.**

*Hon. Sec. Northern District.*—Vacant.

*Returns.*—VII., card 1; form 1; X., 1. Total, 3.

*Hon. Sec. Southern District.*—Acting *pro. tem.*, Henry Barnes, M.D., Portland Square, Carlisle.

*Returns.*, 0.

Total returns from the Branch, 3.

**Cambridge and Huntingdon Branch.**

*Hon. Sec.*—B. Anningson, M.D., Barton Road, Cambridge.

*Returns.*—I., 2; IV., 5; X., 26. Total, 33.

Total returns from the Branch, 33.

**Dublin Branch.**

*President.*—John T. Banks, M.D.

*President Elect.*—Edward Hamilton, M.D.

*Vice-Presidents.*—Loombe Athill, M.D.; Ed. H. Bennett, M.D.

*Hon. Sec.*—R. A. Hayes, M.D., 32, Merrion Square, Dublin.

*Returns.*—I., 8; III., 7; VI., 1. Total, 16.

Total returns from the Branch, 16.

**East Anglian Branch.**

*Hon. Sec. for Norfolk.*—S. H. Burton, M.B., St. Giles Street, Norwich.

*Hon. Sec. for Suffolk.*—W. A. Elliston, M.D., St. Peter's Street, Ipswich.

*Returns.*—Norfolk: VII., 2.

*Returns.*—Suffolk: I., 1; II., 1; III., 1; IV., 5; IVa., 4; VII., 1. Total, 13.

Total returns from the Branch, 15.

**East York and North Lincoln Branch.**

*Hon. Sec.*—E. O. Daly, M.B., 26, Albion Street, Hull.

*Returns.*—IV., 1; IVa., 1; VII., 1. Total, 3.

Total returns from the Branch, 3.

**Edinburgh Branch.**

*Chairman.*—D. J. Brakenridge, M.D.

*Hon. Sec.*—A. Bruce, M.B., 16, Alva Street, Edinburgh.

*Returns.*—I., 6; II., 2; III., 1; VII., cards, 9, forms, 4. Total, 22.

Total returns from the Branch, 22.

**Glasgow and West of Scotland Branch.**

*Convener.*—W. G. Dun, M.D., 2, India Street, Glasgow.

*Returns.*—I., 6; III., 1; VI., forms, 5; X., 1. Total, 13.

Total returns from the Branch, 13.

**Gloucestershire Branch.**

*Hon. Sec. for Cheltenham District.*—E. T. Wilson, M.B., F.R.C.P., Westal, Cheltenham.

*Hon. Sec. for Gloucester District.*—F. T. Bond, M.D., Montpellier Place, Gloucester.

*Returns.*—Gloucester District: I., 3; II., 1; IV., 2; IVa., 1; X., 1; XI., 1. Total, 9.

Total returns from the Branch, 9.

**Lancashire and Cheshire Branch.**

BOLTON DISTRICT.—*Hon. Sec.*—De Vere Hunt, Esq., 46, St. George's Terrace, Bolton.

*Returns.*—I., 53 ; II., 9 ; III., 4 ; IV., 3 ; IVa., 3 ; X., 1 ; XI., 4. Total, 77.

CHESTER DISTRICT.—*Hon. Sec.*—A. Macpherson, M.B., General Hospital, Chester.

*Returns.*—

LIVERPOOL DISTRICT.—*Hon. Sec.*—Creswell Rich, M.B., 4, Canning Street Liverpool.

*Returns.*—I., 29 ; II., 11 ; III., 3 ; IV., 7 ; IVa., 1 ; V., 2 ; VI., 2. Total, 55.

MANCHESTER DISTRICT.—*Hon. Sec.*—J. S. Bury, M.D., 36, Fitzwarren Street, Pendleton, Manchester.

*Returns.*—I., 71 ; II., 12 ; III., 10 ; IV., 4 ; IVa., 3 ; VII., forms, 2 ; XI., 1 Total 103.

Total returns from the Branch, 235.

**Metropolitan Counties Branch.**

*Hon. Sec.*—George Eastes, M.B., 69, Connaught Street, Hyde Park Place, W.

The District Secretaries for the Branch act also as District Secretaries for the Committee.

*Returns.*—I., 67 ; II., 10 ; III., 16 ; IV., 8 ; IVa., 5 ; V., 3 ; VI., 4 ; VII., cards, 6, forms, 11 ; X., 12 ; XI., 6. Total, 148.

Total returns from the Branch, 148.

**Midland Branch.**

DERBY DISTRICT.—*Chairman.*—W. Ogle, M.D.

*Hon. Sec.*—

*Returns.*—I., 5 ; IV., 4 ; IVa., 2 ; VII., form, 1 ; XI., 1. Total, 13.

LEICESTER DISTRICT.—*Hon. Sec.*—W. E. Buck, M.D., Welford, Road, Leicester.

*Returns.*—0.

LINCOLN DISTRICT.—*Hon. Sec.*—C. Harrison, M.D., 30, Newland, Lincoln.

*Returns.*—I., 7 ; II., 11 ; III., 3 ; IV., 4 ; IVa., 2 ; Va., 1 ; VII., forms, 2. Total, 30.

NOTTINGHAM DISTRICT.—*Hon. Sec.*—H. Handford, M.D., General Hospital, Nottingham.

*Returns.*—I., 4 ; II., 6 ; VI., 1. Total, 11.

Total returns from the Branch, 41.

**North of England Branch.**

*Hon. Sec.*—D. Drummond, M.D., Saville Place, Newcastle.

*Returns.*—I., 3 ; II., 3 ; III., 1 ; IV., 1 ; VII., card, 1, forms, 3 ; XI., 1. Total, 13.

Total returns from the Branch, 13.

**North of Ireland.**

*Hon. Sec.*—A. Dempsey, M.D., 26, Clifton Street, Belfast.

*Returns.*—I., 2 ; VII., forms, 2 ; VIII., 1 ; X., 4 ; XI., 2. Total, 11.

Total returns from the Branch, 11.

**North Wales Branch.**

*Hon. Sec.*—W. Jones-Morris, Esq., Portmadoc.

*Returns.*—I., 19 ; II., 2 ; III., 3 ; IV., 1 ; VII., card 1, form, 1. Total, 27.

Total returns from the Branch, 27.

**Northern Counties of Scotland.**

EASTERN DISTRICT.—*Hon. Sec.*—J. W. Norris Mackay, M.D., The Tower, 103, High Street, Elgin.

WESTERN DISTRICT.—*President*, T. Aitken, M.D.

*Hon. Sec.*—Ogilvie Grant, M.B., 35, Church Street, Inverness.

*Returns.*—I., 6 ; II., 1 ; III., 2 ; V., 2 ; VII., form, 1. Total, 12.

Total returns from the Branch, 12.

**Reading Branch.**

*Hon. Sec.*—R. C. Shettle, M.D., 73, London Street, Reading.

*Returns.*—I., 1. Total, 1.

Total returns from the Branch, 1.

**Shropshire and Mid-Wales Branch.**

*Hon. Sec.*—W. H. Packer, M.D., County Asylum, Bicton Heath, Shrewsbury.

*Returns.*—I., 9; II., 1; Va., 1; X., 9. Total, 20.

Total returns from the Branch, 20.

**South-Eastern Branch.**

*EAST KENT DISTRICT.*—*Hon. Sec.*—T. Whitehead Reid, F.R.C.P., 34, St. George's Place, Canterbury.

*Returns.*—I., 44; II., 1; III., 24; IV., 14; IVa., 9; Va., 1; VI., 3; VII., cards, 3; forms, 6; X., 7. Total, 112.

*WEST KENT DISTRICT.*—*Hon. Sec.*—C. Boyce, M.B., 3, Clarendon Place, Maidstone.

*Returns.*—I., 9; II., 2; III., 1; VII., card, 1. Total, 13.

*EAST SURREY DISTRICT.*—*Hon. Sec.*—John H. Galton, M.D., Woodside, Anerley Road, Norwood, S.E.

*Returns.*—I., 9; II., 2; III., 4; IV., 2; IVa., 2; Va., 1; VII., cards, 2; forms, 4. Total, 26.

*WEST SURREY DISTRICT.*—*Hon. Sec.*—T. F. Pearse, M.D., Haslemere.

*Returns.*—I., 9; II., 2; Va., 1; VII., card, 1. Total, 13.

*EAST SUSSEX DISTRICT.*—*Hon. Sec.*—J. C. Uhthoff, M.D., 46, Western Road, Hove, Brighton.

*Returns.*—I., 1; III., 1; IV., 1; IVa., 1; Va., 1. Total, 5.

*WEST SUSSEX DISTRICT.*—*Hon. Sec.*—G. B. Collet, Esq., 5, The Steyne, Worthing.

*Returns.*—0.

Total returns from the Branch, 169.

**South Midland Branch.**

*Hon. Sec.*—G. Percival, M.B., Northampton.

*Returns.*—I., 2; II., 2. Total, 4.

Total returns from the Branch, 4.

**South of Ireland Branch.**

*Hon. Sec.*—T. Gelston Atkins, M.D., 17, St. Patrick's Hill, Cork.

*Returns.*—I., 3; VII., forms, 2. Total, 5.

Total returns from the Branch, 5.

**South Wales and Monmouthshire Branch.**

*Hon. Secs.*—D. A. Davies, M.B., De la Beche Street, Swansea; A. Sheen, M.D., Halswell House, Cardiff.

*Returns.*—I., 31; II., 3; III., 9; IV., 6; V., 1; VII., forms, 10. Total, 60.

Total returns from the Branch, 60.

**South-Western Branch.**

*Hon. Sec. for Cornwall.*—E. Scudamore Angove, Esq., Camborne, Cornwall.

*For North Devon.*—J. E. Square, F.R.C.S., 28, Portland Square, Plymouth.

*For South Devon.*—H. Davy, M.D., Southernhay, Exeter.

*Returns.*—Cornwall: VII., forms, 3. Total, 3.

*Returns.*—North Devon: VII., form, 1. Total 1.

*Returns.*—South Devon: I., 2; VII., card, 1; XI., 1. Total, 4.

Total returns from the Branch, 8.

### Southern Branch.

DORSETSHIRE.—*Hon. Sec.*—C. H. W. Parkinson, Esq., Wimborne Minster.  
*Returns.*—I., 8; II., 6; III., 1; IV., 2; IVa., 2; VI., 1; X., 2; XI., 3.  
 Total, 25.

EAST HANTS.—*Hon. Sec.*—T. C. Langdon, F.R.C.S., Northgate House, Winchester.

*Returns.*—I., 3; III., 3; VII., card, 1; XI., 1. Total, 8.

SOUTH HANTS.—*Hon. Sec.*—Theoph. W. Trend, M.D., 6, Anglesea Place, Southampton.

ISLE OF WIGHT.—*Hon. Sec.*—W. E. Green, Esq., Belgrave House, Sandown, Isle of Wight.

*Returns.*—II., 3; III., 2; IV., 5; IVa., 2. Total, 12.

WILTSHIRE.—*Hon. Sec.*—H. J. Manning, Esq., Laverstock House, Salisbury.

*Returns.*—

Total returns from the Branch, 45.

### Staffordshire Branch.

*Hon. Gen. Sec.*—Vincent Jackson, Esq., Wolverhampton.

*Hon. District Secs.*—East Staffs.: W. G. Lowe, M.D., Burton. Mid-Staffs.: G. Reid, M.B., Stafford. North Staffs.: A. M. McCaldowie, M.D., Stoke. West Staffs.: H. Malet, M.D., Wolverhampton.

*Returns.*—I., 21; II., 4; VII., cards, 2; XI., 1. Total, 27.

Total returns from the Branch, 27.

### Thames Valley Branch.

*Committee and Hon. District Secs.*—C. C. Gibbs, M.D., Surbiton; A. R. Graham, M.B., Weybridge; E. H. Hare, Esq., Kew; N. H. K. Kane, M.D., Kingston Hill; H. H. Murphy, M.D., Twickenham; F. J. Wadd, M.B., Richmond.

*Hon. Gen. Sec.*—F. P. Atkinson, M.D., Surbiton Road, Kingston.

*Returns.*—I., 6; X., 1. Total, 7.

Total returns from the Branch, 7.

### West Somerset Branch.

*Hon. Sec.*—W. M. Kelly, M.D., The Crescent, Taunton.

*Returns.*—I., 5; II., 2; III., 1; VII., form, 1. Total, 9.

Total returns from the Branch, 9.

### Worcestershire and Herefordshire Branch.

*Hon. Sec.*—Geo. W. Crowe, M.D., Shaw Street, Worcester.

*Returns.*—I., 9; II., 1; III., 3. Total, 13.

Total returns from the Branch, 13.<sup>1</sup>

### Yorkshire Branch.

*Hon. Sec.*—A. Jackson, Esq., Wilkinson Street, Sheffield.

*Returns.*—I., 41; II., 15; III., 5; IV., 4; IVa., 2; V., 2; VI., 1; VII., form, 1; XI., 1. Total, 72.

Total returns from the Branch, 72.

### North West Provinces and Oudh Branch, India.

*Hon. Sec.*—S. W. Shirley Deakin, Esq., Allahabad.

*Returns.*—

### Foreign Branch.

H. O. Stuart, Cairo: I., 2; VII., form, 1. J. G. Carageorgiades, Cyprus: VII., card 1. J. Gason, Rome: VII., form, 1. A. Cordes, M.D., Genoa: VII., form, 1. J. B. Richardson, Oudh: III., 2. Brig.-Surgeon J. J. Thompson, Jamaica: IVa., 1. Total, 9.



Returns from members not attached to any branch, or whose branch is not known :—

I.	.	.	.	.	.	.	.	.	.	.	.	.	.	.	.	.	48
II.	.	.	.	.	.	.	.	.	.	.	.	.	.	.	.	.	3
III.	.	.	.	.	.	.	.	.	.	.	.	.	.	.	.	.	7
IV.	.	.	.	.	.	.	.	.	.	.	.	.	.	.	.	.	2
V.	.	.	.	.	.	.	.	.	.	.	.	.	.	.	.	.	2
X.	.	.	.	.	.	.	.	.	.	.	.	.	.	.	.	.	11
<hr/>																	
Total	.	.	.	.	.	.	.	.	.	.	.	.	.	.	.	.	73

Total Returns received during the year from July 1, 1883, to June 30, 1884 :—

I.	Acute Pneumonia . . . . .	580
II.	Chorea . . . . .	122
III.	Acute Rheumatism . . . . .	115
IV.	Diphtheria—Clinical . . . . .	84
IVa.	Diphtheria—Etiological. . . . .	41
V.	Syphilis—Acquired . . . . .	12
Va.	Syphilis—Inherited . . . . .	6
VI.	Acute Gout . . . . .	13
VII.	Puerperal Pyrexia (cards 30, forms 74) . . . . .	104
VIII.	Paroxysmal Hæmoglobinuria . . . . .	1
X.	Habits of Aged Persons. . . . .	78
XI.	Albuminuria in the apparently healthy . . . . .	23
<hr/>		
Total	. . . . .	1179

Puerperal Pyrexia Returns . . . . . 316

[NOTE.]—To keep the lists necessary for such an account as this entails great labour, which would be considerably lessened if Hon. Members would kindly write on their cards, the name of the Branch to which they belong. It is otherwise impossible to avoid occasional mistakes.

## MEMORANDA AND CARDS ALREADY ISSUED.

## MEMORANDUM ON FEVER IN THE PUERPERAL STATE.

*(British Medical Journal, November 24th, 1883.)*

FEVER, in puerperal women, is a condition which every one recognises, but no one understands; and though it is the fashion now-a-days to profess ignorance on every subject, in none is such a profession more honest than in this. Yet there are more men interested to discover the cause and cure of this than of any other sickness, and the inquiry which the Collective Investigation Committee sends out in the *British Medical Journal* for November 24, 1883, will be both welcome and deserving of the best support that can be given it. The form is simple and easy to fill up, but embraces a wide field, for the Committee, though desirous of obtaining the utmost possible amount of information, surely knows that men busy in general practice cannot make elaborate notes.

The first question is on the predisposing causes. Habits of drink and squalor; ill-health during pregnancy, either mental, as depression, or bodily, such as venereal disease or albuminuria; and local conditions, whether the result of old mischief in the pelvis, or severe injuries in the recent labour, are all said to render women liable to the attack, and it is to some men doubtful whether bad lacerations in unhealthy women may not of themselves take on malignant inflammation, and the poison of the fever be gotten in the patient's system, unconnected with contagion from without. There will be useful hints gained from the replies to this first question; but the second and third will, if well answered, be of the highest value. Puerperal fever differs from all other diseases in this, that its claim to be considered a separate disease at all is disputed by some of the best writers, who base their objections both on the causes which excite it, and the symptoms which it shows. It has been traced to at least three different families of infection—specific fevers, septic poison, and sewage matter. Instances are told of its arising from contact with typhus, scarlet fever, measles, and small-pox; and while some believe that all the scarlatinal cases are really cases of septicaemia with a rash, others hold that many are called puerperal which are really specific fevers. Facts would be of the greatest importance which should bear on this point, either by showing that another person caught a specific fever from what seemed a case of puerperal pyrexia, or that the course of a specific fever was modified when occurring in the puerperal state.

Septic poison, again—the sort of poison, that is, which produces erysipelas, diffuse cellulitis, or pyæmia, in a surgical wound, for a more exact definition is yet impossible—is the favourite cause with some medical writers. Of this kind are those attacks which have been traced to overcrowding in hospitals, and to infection from a case of these diseases or from a dead body, especially where death was caused by any illness of the same character. Sore-throat or pneumonia, occurring in the same house, may again suggest endemic poison. Thirdly, sewage poison, foul smells, bad drains, ashpits, and other sorts of filth, have been often held to blame, and, since a recent royal instance have come still more prominently into notice. With this diversity of cause varying symptoms would be but natural

and the leading points of difference are put in the *third* question of the form. Some say that a rash of peculiar position and character marks a distinct pyæmic variety; while others maintain that many of these cases are scarlatina—modified, perhaps, by the puerperal condition. Sore-throat raises the suspicion both of scarlatina and diphtheria; and the presence or absence of membrane will, in this connection, be of great importance. In certain cases severe abdominal symptoms show the presence of general peritonitis; others, again, occur where the stress of the disease is confined within the pelvis; and it sometimes happens that several patients are thought to have mere local non-infecting peritonitis, until the series is closed by an unmistakable attack of puerperal fever, and the doctor learns too late the real character of the former cases. The local inflammations elsewhere include that of the breasts (whose condition is always important), phlebitis, and the scattered abscesses, pneumonia, pleurisy, pericarditis, and suppurating joints of pyæmia. The womb is often large and tender, and frequently appears, on *post mortem* examination, to have been the starting point of infective inflammation. Lastly, the history of the symptoms will not only be itself of interest, but, since the disease is one in which treatments the most diverse have without doubt found a great success, should throw much light on that most pressing question, What plans are best adapted to the several types of the attack?

After looking through this form, it cannot but be felt that a better opportunity and a fitter subject for collecting the general opinion of medical men has seldom been offered. There is not a question which does not aim at points on which the ablest differ, nor one which is not in the power of the busiest to answer; and it is to be confidently hoped that the returns may make a turning point in the history of the disease.

## No. VII.

**PUERPERAL PYREXIA:** *with regard to its (1) dependence upon (a) other prevalent diseases, and the channel of production therefrom, (b) defective sanitary conditions, or (c) self-infection; (2) symptoms; (3) treatment; (4) results.*

(Reply, where possible, by erasing words on card.)

Observer's Name\* .....  
Address\* .....  
Nurse's or Midwife's Name\* .....  
Address\* .....

\* These particulars may be suppressed if so desired by the observer. The card must, in that case, when sent to the Secretary of the Collective Investigation Committee, 161A, Strand, W.C., be accompanied by a sheet of paper on which these names and addresses are written. Such information will be regarded as strictly confidential.

*This card, as soon as filled up, to be returned to the Secretary of the Collective Investigation Committee, 161A, Strand, London, W.C.*

Initials of patient. Married, single, widow.  
Age.

Previous health—good, bad, indifferent.  
Number of children at full time (a) born alive.

(b) stillborn. Premature.  
Were any recoveries retarded?  
Were any recoveries attended by pelvic complications?

Occupation. and of husband.  
Temperate, intemperate, total abstainer.  
Food sufficient, insufficient.

Washing done at home or out? Mangling at home or out?

Was patient a recipient of a lying-in bag?  
Residence a week before and at confinement—country, town, in a row, detached, semi-detached, in a public institution.

Sanitary condition of house—good, bad, indifferent.

Sanitary condition of lying-in room—good, bad, indifferent, near drain, cesspool, water-closet, bath-room.

INFECTIOUS DISEASES TO WHICH SHE MAY HAVE BEEN EXPOSED.

Nature of disease.

Date of exposure.

Place of exposure—in same house, neighbouring houses, elsewhere.

If the medical attendant has recently visited any cases of an infectious or septic nature, state

Disease Date.

Or if he has recently made any *post-mortem*, state

Disease Date.

State precautions adopted to prevent diffusion of disease.

Has nurse or midwife recently attended any patient with pyrexia or other suspicious illness after confinement?

HEALTH OF PATIENT DURING PREGNANCY.

Bodily health good, fair, bad.

Mental condition—normal, depressed.

Anasarca—much, little, none.

Urine—not examined, normal, albuminous.

Veneral disease—old, recent, present.

PRESENT CONFINEMENT. Date.

Atmospheric condition—dry, damp, wet, cold, mild, hot.

At full term or not?

Duration of first stage of labour.

Duration of second stage.

Natural or instrumental delivery.

Expulsive pains severe, natural, feeble.

Hæmorrhage severe, moderate, slight.

Sex of child. M. F. Weight.

Living, stillborn, putrid.

Abnormalities during labour, state details.



Laceration of uterus, vagina, perineum, labia—severe, slight, none.  
DELIVERY OF PLACENTA—Interval after birth of child.

Natural or manual.

Placenta—torn, whole, sweet, foul. Uterus fully contracted, relaxed.

Retention of clots or membranes?

Drugs given—chloroform, ergot, opium, or

Pulse-rate before birth within one hour.

Temperature before birth within one hour.

PROGRESS AFTER CONFINEMENT UNTIL ONSET OF PYREXIA.

Highest pulse-rate noted. Lowest.

Highest temperature noted. Lowest.

After-pains—severe, moderate, slight.

Sleep—excessive, normal, deficient.

Appetite—good, fair, bad. Diet.

Tongue—moist, dry, clean, coated. Bowels—regular, loose, costive.

Abdomen—soft, hard, tender. Uterus—contracted or flaccid.

Micturition—natural, difficult, by catheter.

Urine—normal, albuminous.

Lochia—normal, excessive, deficient, foul.

Sloughing of parts in or near vagina.

Clots passed—many, few, none, offensive, non-offensive.

Antiseptic injections used or not. Uterine, vaginal.

By whom?

Breasts—natural, swollen, tender. Nipple—natural, sore.

Date of first milk. Quantity excessive, moderate, deficient.

PYREXIAL PERIOD.—Date of onset. Duration.

Supposed exciting cause.

Attack preceded, accompanied by rigors? Date of first.

Pyrexia—slight, moderate, severe. Skin—dry, moist, sweaty.

Eruption, if any. Where?

Face—flushed, pale, sallow, jaundiced

Pulse—range of.

Sleep—excessive, normal, deficient. Delirium.

Heart—murmur, systolic, diastolic, apex, base. Pericarditis.

Thrombosis of veins—R. I. iliac, R. L. saphenous, R. L. femoral, R. L. axillary.

Swelling of R. L. leg, of R. L. arm.

Pneumonia—R. apex, base, L. apex, base.

Pleurisy—R. L.

Appetite—good, fair, bad. Bowels—regular, loose, costive.

Tongue—moist, clean, coated, dry, streaked. Vomiting.

Abdomen—tender, tympanitic, flaccid.

Breathing—abdominal, thoracic.

Liver—normal, enlarged, tender. Spleen—normal, enlarged, tender.

Uterus—enlarged, tender.

Lactation—normal, arrested. Breasts—normal, inflamed.

Urine—normal, suppressed, albuminous.

Micturition—natural, difficult, by catheter.

Lochia—normal, excessive, scanty, offensive, suppressed.

Joints.

Other local inflammations, if any. Situation and progress.

TREATMENT—Drugs.

How often.

How long.

Douches—vaginal, uterine.

External applications.

Diet.

RESULT OF CASE—Recovery, partial, complete, death.

Sequelae.

If fatal, account of necropsy. (Additional particulars can be given on a separate sheet.)

Progress of infant during mother's illness.

Was any other person infected from the patient? If so, nature of illness.

## A SHORTER FORM OF INQUIRY.

### No. VII.

FEVER IN THE PUERPERAL STATE: *Will you kindly answer the following questions about the last, or any other, case of Pyrexia after labour of which you have an accurate record?*

Observer's Name .....

Address .....

Date at which case occurred .....

1.—What circumstances may have predisposed to the disease?

2.—Can you trace it to infection from any specific fever, septic influence or other cause?

3.—Was there a rash? If so, where?

What were its characters?

Was there sore throat? If so, what were its characters?

Was there distention of abdomen?

Was there local inflammation in pelvis?

Was there local inflammation elsewhere?

4.—What was the condition of the uterus?

5.—How soon after labour did the fever begin?

6.—What was its duration?

7.—What was the result?

8.—Give a brief history of the course of the disease and the treatment.



## PAROXYSMAL HÆMOGLOBINURIA.

BY STEPHEN MACKENZIE, M.D.

*(On behalf of the Committee).*

THIS affection, although regarded by many as a pathological curiosity, is thought by the Committee to be a proper subject for investigation for the following reasons.

1. In the first place, it is believed that the condition is not so rare as has been supposed; and it is hoped that, when attention is drawn to it, many cases now unrecorded may be brought under observation.

2. We know very little of the life-history of the subjects of this affection, and still less of their family-history. Is it hereditary? Is it specially connected with any diathesis? What are the chances of getting rid of the liability to the attacks as life advances?

3. Finally, records of *post mortem* examinations are much wanted; the only case hitherto published being that by Murri, in which any special alterations were obscured by the presence of advanced visceral syphilis.

Further, the Committee feels that an object which should be kept prominently before it, is the collection of the records of cases of rare diseases, the occurrence of which is so far exceptional that the experience of any one person must be exceedingly limited. Such experience, to be of use as a guide in prognosis and treatment, can only be obtained by collective action. This function of the Committee has been recently dwelt upon by Mr. Hutchinson in his address at Manchester (*British Medical Journal*, November 3, 1883, p. 862). To this end, the Committee is anxious to secure the experience of our hospitals in addition to that of the individual practitioner, and it especially appeals to the resident medical officers and registrars, as well as the physicians and surgeons of the provincial and metropolitan hospitals, to send records of all cases of this disease falling under their observation.

We owe the first description of this affection to Dr. George Harley. As its name implies, it consists essentially in the passage of blood-colouring matter into the urine, in distinction from hæmaturia, in which the blood-corpuscles themselves are present. But this cardinal symptom, which may occur in various conditions (putrid infection, purpura, poisoning by various substances, etc.), in the peculiar attacks which have received the name of paroxysmal hæmoglobinuria, is an idiopathic affection, and is accompanied or preceded by more or less constitutional disturbance: of which, rigor and headache, followed by some rise of temperature, are the most common phenomena. The attack may last from a few hours to a few days.

The colour of the urine varies from that of porter to mere smokiness, according to the period or severity of the attack. It coagulates on boiling, forming a brownish clot. It deposits a copious brown sediment, which, under the microscope, appears to consist of granular matter (broken-down blood-corpuscles?), amorphous urates, oxalates, and granular and hyaline casts.

With guaiacum and ozonic ether, the urine gives the blue tint characteristic of blood-colouring matter. This test is best performed by adding to about a drachm of urine in a test-tube a few drops of fresh tincture of guaiacum and twenty or thirty drops of ozonic ether; the whole to be well shaken, and then allowed to settle, when the ozonic ether rises to the top, holding in solution the red colouring matter of the guaiacum tincture, or, if blood be present, the blue pigment to which this has been transformed. This test should always be used, and suffices, when a spectroscopic examination cannot conveniently be made, together with the absence of blood-corpuscles from the fresh urine, to establish the diagnosis.

With the spectroscope, the urine, which must be filtered and diluted, and then examined in a moderately deep layer, gives the two bands between D and E when oxyhæmoglobin is present, or three characteristic absorption-bands of methæmoglobin, an oxidation-product of hæmoglobin.

The urine should be examined microscopically in every case; and the presence or absence of blood-corpuscles, oxalates, and casts should be noted.

More precise information is needed on the following points. Does albumen appear in the urine before the blood-colouring matter? Does albumen remain, and for how long, after the complete disappearance of the blood-colouring matter?

The subjects of these attacks are usually in fair general health, and many are quite free from any indication of constitutional or organic disease; but a certain number of the reported cases have suffered from ague, or have been exposed to malarial infection. This point should always receive attention, as, in some respects, the attacks have a rough resemblance to a malarial attack; though, undoubtedly, many cases have been quite free from any possibility of such an origin.

In two other cases, there was constitutional syphilis, and these are said to have benefited by antisyphilitic remedies.

It would be very interesting to know whether the liability to these attacks exists in other members of the same family, or is transmitted by descent; or whether other neuroses are common in the relatives of the patient. In one instance, the father and two children have manifested the liability; but this is the only recorded example of heredity.

The influence of cold in causing the attacks has been well established; but it is a point to which attention should be directed, in order to determine, if possible, the kind and degree of temperature which is productive of the attacks.

In a certain number of cases, the first attack has been brought on by a blow, a fall, or by prolonged walking. The cause of the first attack should always be carefully noted. In one case it was associated with an attack of tonsillitis. The influence of muscular exercise is also still obscure, though it appears to favour the onset of an attack. We should also seek to determine whether the alleged exciting cause is always efficient, or whether the paroxysm is only the *occasional* result of apparently similar conditions.

Attention has been drawn to the relation which this disease appears to bear to symmetrical gangrene. This form of gangrene occurs almost invariably in children, and attacks just those parts of the body liable to suffer most from severe cold, as in frost-bite—the tips of the fingers, toes, ears and nose. The circulation is very feeble; and the formation of gangrene is preceded by redness, followed by lividity, of the affected parts. It has been observed that such patients frequently pass bloody urine, which deposits oxalates in abundance.

There are very considerable differences in the accounts of the symptoms observed during these attacks. The temperature may be normal, or even subnormal, or may be as high as 103° or 105° Fahr. The skin may be dry, or covered with perspiration; cutis anserina and urticaria have been observed; duskiness of the skin and conjunctiva, or actual jaundice, may be present. The spleen is often, but not always, enlarged. The liver may be tender, or enlarged, or quite normal.

According to Hayem, the blood, under the microscope, presents nothing very special, only the appearance of slight anæmia; but the serum contains free hæmoglobin. Whenever it is possible, a specimen of serum should be obtained by venesection or blistering, and submitted to spectroscopic examination.

It has been noticed that the urine during the intervals may contain albumen, indican, etc. This persistence of albumen is of much practical importance, and information is specially asked for on this head. Whenever albumen is found, the presence or absence of casts, and their characters, should be noted.

Treatment during the attacks has been mainly symptomatic—rest in bed, warmth, etc. The late Dr. Warburton Begbie considered that he had cured a case with scruple doses of chloride of ammonium; but the duration of the attack is so variable, that it is possible to be deceived as to the value of a remedy. In other hands, chloride of ammonium has done no good; while the majority of observers speak favourably of quinine in relatively large doses—ten grains three or four times a day. The marked benefit ascribed to antisyphilitic treatment in two cases has been already referred to.

Dr. C. A. McMunn, of Oakleigh, Wolverhampton, has kindly consented to examine spectroscopically any fluids sent to him in connection with this inquiry. Packages should contain the observer's name and address, to whom Dr. McMunn will send his report.

*The Committee is indebted to Dr. Stephen Mackenzie and Dr. Robert Saundby for this memorandum, and also for the card of inquiry which they have prepared.*

## No. VIII.

PAROXYSMAL HÆMOGLOBINURIA:—*Synonyms.*—*Intermittent Hæmaturia, Intermittent Hæmatinuria.*

Observer's Name .....  
Address .....  
Date of last obs. ....  
(Reply where possible by erasing words on paper.)

(When in doubt refer to accompanying memorandum paper for explanation.)

(Please return this paper when filled up to the Secretary, Collective Investigation Committee, 161a, Strand, W.C.)

Initials of patient—or case number.

M. or F. Age.

Single. Married. Widowed. Occupation.

History of similar affection in relatives (specify relative, age at which it occurred, and issue of case if possible).

Other hereditary diseases in family.

## PRE-PAROXYSMAL CONDITIONS.

Locality of residences.

Are any of these reputed aguish?

HAS PATIENT EVER SUFFERED FROM AGUE?  
If so state—

Place where it was contracted.

Form. Duration. Date.

OTHER ANTECEDENTS (with dates).

Scarlet Fever. Syphilis. Bright's Disease.

Injury of any kind. Nature and date.

If urine has been examined before attack state characters.

Mention any minor ailments to which patient is liable.

Date of first known paroxysm.

Exciting cause of first attack.

Exciting cause of subsequent attacks.

PAROXYSM. Date.

Accompanied by rigor, chilliness, cutis anserina, urticaria.

Temperature.

URINE. How soon after initial symptom blood-colouring matter appeared in the urine.

NATURE OF COLOURING MATTER.

Naked eye appearances.

By guaiacum test.

By spectroscope.

MICROSCOPIC EXAMINATION. Blood-corpuscles, Blood-crystals, Oxalates.

Cast (nature).

Other Deposits.

ALBUMEN. Prior to colouring matter, with the colouring matter, subsequent to colouring matter.

Quantity of urine in 24 hours.

Specific gravity. Before attack , during , after ,

Reaction. Before attack , during , after ,

Presence of indican. Before attack , during , after ,

ENLARGEMENT OF SPLEEN. Distinct, doubtful.

STATE OF BOWELS. Regular, constipation diarrhoea.

SKIN. Perspiration, general, local, absent.

COLOUR of Skin, of conjunctiva.

EXAMINATION OF BLOOD.

Character of coloured blood-corpuscles.

Do. of colourless.

Serum. Coloured, uncoloured, spectroscopic examination.\*

SUBSIDENCE OF ATTACK. Spontaneous, due to treatment (specify nature).

## POST- OR INTER-PAROXYSMAL CONDITIONS.

URINE. Sp. gr. Reaction. Colour.  
Guaiacum test. Albumen.

Deposits (nature of).

ENLARGEMENT OF SPLEEN.

ENLARGEMENT OF LIVER.

COLOUR OF SKIN. Conjunctiva.

Relapses, frequency of.

COMPLICATIONS AND ASSOCIATIONS.

Symmetrical gangrene.

Paralysis (nature).

Nephritic or vesical calculus.

Chilblains (where situated).

Have you had a case of this disease under observation at any previous date?

If so, what was the progress and issue of the case.

\* Serum may be conveniently obtained by a blister or cupping. Serum and urine may be sent to Dr. C. A. McMunn, Wolverhampton, who will examine it spectroscopically and report on specimen.

## No. IX.

## ENTERIC FEVER.

*This card is by order of the Committee withdrawn for further consideration.*



## AN INQUIRY CONCERNING PERSONS WHO HAVE ATTAINED OR PASSED THE AGE OF EIGHTY YEARS.

MEMORANDUM BY PROFESSOR HUMPHRY.

*(On Behalf of the Committee.)*

THIS inquiry, as will be seen by the card, is intended to be general, the object being to obtain by Collective Investigation on a large scale, information respecting the present and past condition, habits, and maladies, as well as the family history and other circumstances, of those who have attained to advanced periods of life, in order that we may be able to ascertain, with greater certainty than we now can, what are the circumstances which favour longevity, the means by which it may be promoted, and the maladies which are most, and those which are least incidental to it.

The following are some of the questions which arise in connection with this subject, and for answers to which we may look.

What bodily conformation, temperament, and habits, are most associated with, or conducive to, longevity?

Do women more frequently attain to great age than men, and have women somewhat below the ordinary stature the advantage in this respect?

Are the married or the unmarried, the stout or the spare, the active or the sedentary, the industrious or the idle, the indoor students or the outdoor workers, the well-to-do or the poor, the town-dwellers or the country-dwellers, the more likely to become octogenarians?

It is said that "small eaters and short sleepers are long livers." Is this so? Will the "early to bed and early to rise" maxim receive confirmation? What is the influence of alcohol?

It has been remarked that a considerable proportion of aged persons have been more or less ailing during a great part, or the whole, of their lives. Is that the case? It has also been remarked that many of them have been troubled with constipation, and that many have long been in the habit of resorting to aperient medicine.

The cartilages of the ribs and the trachea have been found soft and elastic in some very aged people, Old Parr forming no exception in this. Should this be shown to be generally the case, the inference would follow that persons in whom they are not so are not destined to attain to great age.

Do octogenarians often suffer, or do they enjoy a comparative immunity, from affections of the urinary and genital organs, and of the abdominal organs, also from malignant disease and scrofula?

Are they on the whole comparatively exempt from disease?

To what affections are they most liable, and to what morbid influences are they most susceptible? Do any maladies seem to have an influence in promoting longevity? What influence upon the longevity of an individual has the age of the parents at his birth? Do twins or the children of twins often attain great age?

Information, though not positive, yet of much interest and importance, upon these and other points, will accrue from the replies to the questions on the forms. It need hardly be said that the questions are by no means exhaustive, and that information upon other points which are judged to be of interest and importance by those who fill up the forms will be valuable, as also information on any special points in particular cases which seem worthy of note.



Though the questions are such that they may for the most part be answered by the persons themselves, or by their friends, it is hoped that, in most instances, the observations will be made and the information given by medical men; and the person who fills up the form is in each instance requested to state whether he is a medical man or not.

It will be an additional advantage if some information can be gleaned respecting the succession of maladies in the same person, and in different individuals of the same family, or respecting the preservative influence upon the system of certain maladies against the inroads of others.

Something in the Hereditary Problem may be also learned respecting the cross action and modifying influences of certain diseases. For instance, is there any foundation for the view that chronic gouty affections retard the development of other diseases?

The strength and enduring quality of the body, like that of a chain, must be measured at its weakest point; and though in it, more than in a chain, the strength and quality of some parts may compensate for deficiency in others, yet the very opposite may be the result. The stronger organs may relieve, but they may also oppress, the weaker members. A strong digestive system may overload a weakly circulation, and prove injurious to the liver, lungs, or kidneys, in fact be a disturbing agent to the general nutrition. The requisite for longevity, therefore, we may expect to be not so much strength of organs as their enduring quality, their good mutual adjustment, in other words, their good balance. The replies relating to "plethora" and other features of general condition will have an important bearing on this view.

## No. X.

## AGED PERSONS.

*An Inquiry concerning the general condition, habits, and circumstances, past and present, and the Family History of PERSONS who have attained or passed the AGE of EIGHTY YEARS.*

Name of Informant .....  
Address.....  
Date .....

When the information relates to a person recently deceased, the date and cause of death should be given, if they can be ascertained.

*The replies to this inquiry will be most valuable when given by a Medical Man; but the questions have been so arranged, that, with the exception of some on the last page, they may be answered by another person. Partial information will be gladly received. When filled up, the form should be returned to—*

The SECRETARY of the  
COLLECTIVE INVESTIGATION COMMITTEE,  
161A, Straud, W.C.  
*who will supply additional copies if desired.*

N.B.—*Reply when possible by erasing words on the form if the reply is negative, and by drawing a line underneath if it is affirmative; i.e., draw the pen through the words to which the answer is "No," and underline those to which it is "Yes."*

## CONDITION AT THE PRESENT TIME.

Name or Initials.  
Age. Male or female. Single, married, widowed.  
Residence Occupation.  
Circumstances.—Affluent, comfortable, poor.  
GENERAL CONDITION.

Fat, spare, average; full-blooded, pale, average; strong, feeble, average.

Height feet inches. Weight

Figure.—Erect, bent.

VOICE.—Loud, clear, full, weak.

SIGHT.—Good, short, long. Are glasses required for reading? If so, for how many years?

HEARING.—Good, bad, indifferent.

Are the joints of the fingers or hands natural, stiff, or deformed?

TEETH.—How many remaining? Artificial teeth used? If so, how long?

DIGESTION.—Good, bad, moderate.

Appetite.—Good, bad, moderate.

Small eater, large eater, moderate. How many meals each day?

Amount and kind of alcoholic beverages daily?

Amount and kind of other beverages daily?

Amount of animal food daily?

Bowels.—Act daily, alternate days, irregularly. Are aperient medicines taken frequently, rarely?

DISPOSITION.—Placid, irritable, energetic, lethargic.

INTELLECTUAL POWERS.—High, low, average. Give any details.

Memory.—Good, bad, moderate, for past or recent events.

HABITS.—Active, sedentary, confined to bed. Amount and kind of outdoor exercise.

Smokes tobacco.—Much, little, moderately. Takes snuff.

Sleep.—Good, bad, moderate. No. of hours. Hour of going to bed.

Of rising.

ANY PRESENT MALADIES.—Their nature and duration.

State any other points in the general condition, habits, etc., worthy of mention.

## PAST HISTORY.

Occupation.  
Residences.  
Age when married. Duration of married life. No. of children.  
*Circumstances.*—Affluent, comfortable, poor.  
First, second, third, or child of parents.  
*GENERAL CONDITION.*—Stout, spare, average; delicate, robust, average; health usually good, moderate, often ailing, rarely ailing.  
*DIGESTION.*—Usually good, indifferent.  
*Bowels.*—Usual condition of.  
*Baldness or greyiness* of hair; occurring early in life, or late.  
*DISPOSITION.*—Placid, irritable, energetic, lethargic.  
*INTELLECTUAL POWERS.*—High, low, average. Any special evidence respecting them.  
*HABITS.*—Active, sedentary, moderate. Amount and kind of outdoor exercise.  
Hours in bed, hour of rising.  
Good, bad, average sleeper.  
*APPETITE.*—Good, indifferent. Large, small, average eater.  
Amount and kind of alcoholic beverages daily.  
Amount and kind of other beverages daily.  
Amount of animal food daily.  
*Smoked tobacco.*—Much, little, moderately.  
*Has taken snuff.*  
*ILLNESSES UNDERGONE.*—Their nature, and the period at which they occurred. Whether severe. Their duration and completeness of recovery.  
*SLIGHT AILMENTS.*—To which specially liable; and period of life at which they occurred.  
*ACCIDENTS.*—With dates.  
State any other points or peculiarities which may seem worthy of mention, in the case of either sex.

## FAMILY HISTORY.

Ages of brothers now alive  
" sisters "  
" children "  
Was there any, and if any, what, blood-relationship between father and mother, or between grandparents on either side?  
Age of father when the subject of the inquiry was born.

Age of mother when the subject of the inquiry was born.

Is any member of the family known to have had cancer, consumption, scrofula, gout, rheumatism, epilepsy, or insanity? State which member in each case.

State any other points in family history that may seem worthy of mention.

	AGE AT DEATH.	CAUSE OF DEATH.
Father's father ..		
" mother ..		
Mother's father ..		
" mother ..		
Father .....		
Mother .....		
Brothers .....		
Sisters .....		
Sons .....		
Daughters .....		

*The following questions can only be answered by a Medical Man.*

CHEST-GIRTH round nipples in inspiration, inches.

CHEST-GIRTH round nipples in expiration, inches.

ELASTICITY of rib-cartilages (*as ascertained by gentle pressure upon them, and upon lower end of sternum*), distinct, indistinct.

PULSE.—No. per minute, regular, intermitting, large, small, compressible, incompressible. Arteries tortuous, visible, even, knotty.

RESPIRATIONS.—No. per minute, regular, irregular.

ARCUS SENILIS.—Much, little, absent.

TEETH.—How many remaining?

Upper incisors, canine, molar.  
Lower "

ANY EVIDENCE OF FAILURE of heart, lungs, brain, urinary or other organs?

MICTURITION.—Slow, difficult, painful, natural.

## No. XI.

## ALBUMINURIA IN APPARENTLY HEALTHY PERSONS.

The importance of albuminuria, and its value as a symptom of disease of the kidneys, has recently been the subject of discussion. After the observations of Bright had demonstrated the frequency with which kidney-disease was associated with albuminuria, it came to be generally accepted that all persons whose urine was albuminous were suffering from disease of the kidneys, with the exception of those in whom a sufficient cause for it was found in some temporary condition, such as fever, or in heart-disease. To this view some exception must now be taken; for we find that, although most persons suffering from disease of the kidneys have albumen in the urine, yet cases of albuminuria are met with in which there are no other indications of kidney disease. It is needless to adduce examples of the latter condition, about which so much has been written during the last few years. Some writers have gone so far as to speak of "physiological albuminuria," and to say that an exceedingly small trace of albumen is constantly present in healthy urine. It is possible that this may be so; but this need not trouble the physician, for the ordinary clinical tests fail to detect it. It has, however, been stated that albumen, in easily appreciable quantity, can be detected

in the urine of a certain number of reputedly healthy persons. The question now presents itself, are these persons sound? Can their lives be regarded as safe for insurance or other purposes for which a certificate of good health is required? Or do we possess in this symptom a valuable indication, forwarning us of approaching organic disease? It ought not now to be difficult to collect sufficient evidence upon which to found satisfactory answers to this question. The inquiries on the following page are submitted to the profession by the Collective Investigation Committee, with the view of obtaining a large body of evidence upon this matter, which will represent the combined experience of many observers.

*As the Committee hope to be able to report on this subject without delay, the annexed form should be filled up as soon as possible, and returned to "The Secretary of the Collective Investigation Committee, 161a, Strand, W.C.," who will gladly forward as many additional forms for other cases as may be required.*

*Observer's Name,  
Professional Titles,  
Address,*

#### DETAILS OF THE SUBSEQUENT HISTORY OF A CASE IN WHICH ALBUMINURIA OCCURRED IN AN APPARENTLY HEALTHY PERSON.

Initials. Sex. Occupation. Age.

Age at which albumen was first discovered in the urine.

What circumstances led to its detection?

By what tests was its presence demonstrated?

What was its proximate amount?

Duration of albuminuria?

Was it constant or intermittent?

If intermittent, what circumstances (if any) appeared to determine or favour its appearance?

Was the person robust or delicate?

Was the general health undoubtedly good?

Has it always been so? Have there been other ailments?

If so, what were they?

Was the individual of active or sedentary habits?

If a female—had she previously borne children? If so, how many?

Could the albuminuria be traced in any way to pregnancy?

Was the person addicted to any injurious habit?

Did the albuminuria bear any relation to food, to exposure, to cold, or to bathing?

Is there any family history of gout or of Bright's disease?

Has the person suffered from gout?

Had the patient previously suffered from scarlet fever?

If so, at what age? Was it followed by albuminuria?

WHAT WAS THE SUBSEQUENT HISTORY OF THE CASE? (Note especially the nature of any illnesses, whether at any time there was dropsy, or any other symptom of Bright's disease, and the cause of death if it has occurred.)

If the subject remained in good health, state the length of time he was under observation.









V. E F  
1883







